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URETHROPLASTY*

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This paper deals with stricture of the urethra, as well as a break in continuity of the urethra from trauma or inflammatory slough, and also with the treatment of stricture of the urethra, as well as repairing the break in continuity of the urethra.

I graduated from medical school in 1916. At that time external urethrotomy was taught as the treatment for urethral obstruction. Since that time, I have seen very little in the literature about its treatment or about perineal surgery. It is my opinion that teaching of perineal surgery is neglected to a large extent. While it is true that urethral obstruction is relieved by external urethrotomy, this obviously produces more scar tissue and more obstruction, and calls for another urethrotomy in a short time. Several years ago there was a wave of advocating opening the stricture with the electric urethrotome; this of course produced much more scar tissue and only lasted for a short time, so it naturally soon fell into disrepute.

The continuity of the urethra is lost either by scar tissue, slowly but progressively forming a contracture until obliteration is brought about, or the continuity is destroyed by trauma, accidental destruction or, it is quite possible in some cases, it is destroyed by the surgeon dissecting it away without recognition.

Strictures should always be recognized early and dilated over a long period of time to prevent excessive scar tissue forming, but it is so insidious in its formation that it is most frequently overlooked by both patient and doctor until irreparable damage is done to the entire urinary tract.

CASE NO. 1

A picture of urogram will be shown in the presentation. A male, age 59, was seen critically ill with dysuria, uremia, and infected urine. Examination revealed an annular stricture in the mid portion of the urethra. The urethra was well dilated above the stricture, and a marked fibrous bar was found at the bladder neck, marked diverticulosis of the bladder, a dilated right ureter and pelvis with poor function; the left ureter was displaced by a diverticulum. The left kidney was functionless and not demonstrable. All of this was continuously overlooked at previous examinations. This could be avoided if all the physicians that attempted to make a complete examination had available some acorn sounds to pass down the urethra and determine if there is narrowing of the lumen. Since the advent of penicillin and sulfonamides, the number of new cases of urethral stricture in the male will be greatly reduced, but we will always have urethral obstruction and destruction from one cause or another, so it behooves us to keep abreast with the management of such conditions.

As long as the continuity of the urethra is intact, and a filiform sound can be passed, continuous dilatation is the treatment to be instituted and is the only treatment that should be used. An external urethrotomy is contraindicated and is harmful. If there are fistulae and multiple

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This paper was illustrated by moving picture films, and without the films the paper is somewhat incomplete, but gives the reader to some extent the impression of what the possibilities are in perineal surgery.

strictures, and it is impossible to get a filiform through, you are then justified in doing an external urethrotomy at the point of the most proximal fistulous opening, then retrograde dilatation will sometimes re-establish continuity in the urethra; this is a process that will have to continue during the lifetime of the patient.

If there is a fistula through the perineum and the urine passes through the fistula and the urethra is intact from there outward, the procedure of choice is dissecting the fistula down to the stricture, removing all the scar area and then freeing up both the proximal and distal stumps of the urethra until there is enough free and loose pliable urethra to be brought together and anastomosed end to end. A good deep guy suture should be placed in good tissue above and below and on either side of the repair to prevent any tension appearing at the point of anastomosis. (This will be illustrated in the presentation). This is the most common stricture where there is a complete break in the continuity of the urethra, and is the most satisfactory one to repair as it requires less dilatation, if any, in the future.

CASE NO. 2

A veterinarian, 60 years of age, had been passing most of the urine through the perineum for quite some time, but now having difficulty he applies for relief. A repair by dissection, and end-to-end anastomosis gave him complete comfort. He has been dilated about once annually since that time. This is by far the best type of treatment in the individual who yet has his sexual power.

There is another type of stricture in the person past middle age who has a destruction of the membranous urethra down to the prostatic portion and outward to within the scrotum, also in a person who has a long penis (making more urethra available). This case may have all the urethral bed dissected free of infected tissue and all of the scar removed. Then the corpus is dissected from its attachment on the rami of the pubes and pulled backward through the skin and soft tissue, dissecting the urethra away from the shaft of the penis. As much of the shaft of the penis is sacrificed as is necessary to span the good urethra along the perineum and to the prostatic portion for anastomosis. The stump of the penis is bifurcated and re-attached to the rami of the pubes at its original location. Dorsal penis artery and vein are sacrificed, as well as the sexual power. However, this gives good functional results and requires very little dilatation in the future.

CASE NO. 3

This patient was a male, aged 45 years, with gangrenous posterior urethra from stricture and early extravasation. The perineum was opened, all the urethra dissected from the prostatic portion well into the scrotum, the base of the penis dissected from the rami of the pubes, the artery and vein were ligated. The penis was pulled backward, urethra detached from the shaft and enough of the shaft of the penis was sacrificed to enable the urethra to extend back to the prostatic portion. The shaft of the penis was bifurcated and reattached to its original location, and the urethral stump anastomosed to the prostatic urethra. Urine was drained by an indwelling catheter, and wound closed in the usual manner. Collateral circulation was re-established and the penis lived as well as the urethral attachment. This has been about eight years and the individual has required very little dilatation. He has no sexual power, but has a healthy urinary tract.

There is another type of break in the continuity from either stricture or trauma found in the young individual or in one who is quite active and who does not have a lengthy urethra. This type may be repaired by the skin tube graft taken from the axilla. This procedure will be shown by a moving film in this presentation, also a picture of a case to show the end results of skin tube transplantation.

CASE NO. 4

A boy, 13 years of age, was crushed in an automobile accident, rupturing the posterior urethra. He was carried to a hospital and had a suprapubic cystotomy. A few days later an external urethrotomy was performed with a good deal of difficulty. Evidently the urethra was not clearly recognized, but an indwelling catheter was left in for drainage, and the supra-pubic tube was removed. Healing took place and the catheter was removed from the urethra. He voided urine through the urethra for a few days, then obstructed again. At this time, the urine broke into the rectum, forming a recto-urethral fistula. Atresia of the fistula took place gradually, then it was difficult to pass the urine inot the rectum. At this time some other procedure had to be done. I was asked to see the patient, and

selected a skin tube transplantation, which was carried out according to the following technic.

The perineum was opened and dissected to good urethra anteriorly, and scar tissue removed posteriorly to the fistula. This was carried down to the prostatic urethra, which was isolated and freed up, and the fistula dissected back to the rectal wall. At this point, the anal sphincter was severed, the mucous membrane was brought forward and closed, and the sphincter approximated and repaired. The perineum was built up between the rectal wall and the new urethral bed, a skin tube was taken from the axilla and transferred to the urethral bed. A catheter was placed through the penis and tube, into the bladder. One end of the tube was anastomosed to the prostatic urethra and the distal end was anastomosed to the urethral stump. All of this tissue was bathed with penicillin solution. The wound was closed over the tube lightly to prevent pressure and a retention catheter was left in for three weeks. At this time, the catheter came out and he voided well, but it was placed back at each two-day interval, and at one of the procedures, the catheter was pushed back into the rectum and this re-established the recto-urethral fistula. There was so much infiltration of tissue at this area, it was thought best to side-track the fecal matter by colostomy. At the present time, he is wearing a urethral catheter, and efforts to close the fistula are being made.

In conclusion, I might say we physicians owe a great deal to the advent of penicillin and sulfonamides, which enable us to do this type of work and get primary healing.

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CALCAREOUS DISEASES OF THE PANCREAS*

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NEW ORLEANS

About one year or more ago, while I was serving with a General Hospital in Italy, B. Y., a white male 38 years of age was admitted to my ward with the chief complaint of chronic diarrhea. He dated his illness back to 1938, at which time he was suddenly seized with a severe, excruciating pain in the upper right quadrant of his abdomen. Nausea and vomiting were marked. He was seen by his physician who found his abdomen rigid and who made a diagnosis of possible appendicitis. Large and frequent doses of opiates were necessary for relief. Within four or five days, however, he had sufficiently recovered to undergo a gastrointestinal series. The findings were negative. The diagnosis was made of "colon trouble".

Following this attack, he developed an intermittent diarrhea. This was aggravated by dietary indiscretions and alcoholic indulgences. His stools were frequent, containing some mucous but no blood. Occasionally pain in the upper abdomen was experienced, but it was always relieved by a few doses of codeine.

When I saw this man, he had served in the Army for four years. In the States, he had very little trouble except that he had noticed some digestive disturbance after heavy and greasy meals. Usually he took no medication, but at other times he would resort to a few doses of paregoric. For 24 months this soldier had been on foreign soil. After landing in Africa, he, along with 60,000 or more comrades, had a few "spells" of diarrhea, but he, unlike many of the others, was able to control it with his own medication.

During the ensuing year and a half, he had fought in many battles, receiving three wounds, and had been hospitalized a fourth time for malaria. He was admittedly fatigued by December 1944, when his diarrhea became very intense. He passed between five and 20 bowel movements a day. They were light yellow to dark brown in color, always soft to mushy in consistency and contained varying amounts of mucous. He had lost some 15 pounds in two or three months and was complaining of weakness, anorexia and excessive nervousness when I first saw him.

His past history was significant in that he admitted drinking heavily at times. This began at age 20, but he vehemently denied any chronicity.

The family history was essentially negative except that his mother died at 51 of an unknown stomach ailment. His father died at 63 of kidney trouble. He had five brothers and two sisters living and well. There was no history of diabetes or other metabolic disturbances

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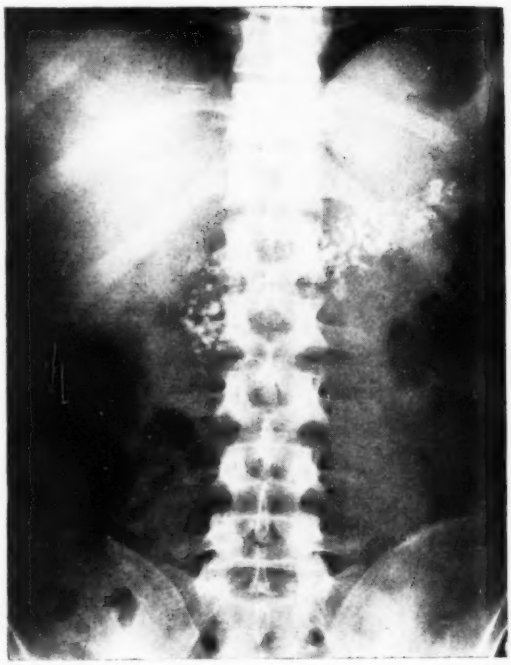
The physical examination revealed a well developed and fairly well nourished male adult, not acutely ill. There was no evidence of any great loss of weight. There was some slight fullness and tenderness in the upper right quadrant, but otherwise there were no positive findings. Routine proctoscopic examination revealed a normal mucous membrane. There was a slight excess of mucous present.



Fecal examinations were negative for ova and parasites, and the cultures were negative for pathogens. On regular diet there was reported, "very much undigested food, moderate amount of fat, neutral fats and fatty acid crystals."

A barium enema was performed with the report negative so far as the colon was concerned, but "numerous small discrete, dense shadows probably calcification, were noted throughout the whole of the pancreas." (Slide 1.) A subsequent flat plate

more definitely identified the calcification and confirmed the original impression. (Slide 2.)

Frankly, I had never seen a picture such as this. A gastrointestinal series was then taken further to demonstrate the relationship of this group of calcifications to the stomach and duodenum. (Slide 3.) This, finally and conclusively, was a case of disseminated calcification of the pancreas.

There were many questions which immediately entered my mind. Unfortunately, our library facilities were meager and my scramble into the literature left me anything but satisfied. Consequently, with the aid of our biochemistry and x-ray depart-

ments, my investigations were begun with the following results.

LABORATORY FINDINGS

Urinalysis—repeatedly negative for sugar, albumin and microscopic elements. Urinary amylase 100 units (normal).

Fecal studies: (1) Ova, and cysts and parasites—negative; (2) bacteriology—negative for pathogens; (3) food content: (a) On regular diet—very much undigested food, moderate amount of fat, neutral fats and fatty acid crystals; (b) on Schmidt diet—(carbohydrate 200 grams, protein 100 grams, fat 100 grams)—no undigested meat fibers; no excess starch granules, no excess fat—quantitative 13.7 per cent (normal 15-25 per cent).

Gastric analysis: free HC1—66°; total acidity— 94°.

Blood studies: (1) Hematology: red blood cells 5,250,000, hemoglobin 95, white blood cells 11,250, neutrophiles 55, lymphocytes 40, monocytes 3, eosinophiles 2.

(2) Chemistry:

		Normal
N. P. N.	33.3 mg	25-35
Chlorides	473 mg.	350-500
Cholesterol	235 mg.	140-250
Amylase	124 units	70-200
Calcium	11.3 mg.	8.5-11.5
Phosphorous	3.8 mg.	3-4.5

(3) Glucose Tolerance Test

Fasting—115 mg.
30 min.—196 mg.
1 hr. —224 mg.
2 hr. —152 mg.
3 hr. —115 mg.

A typical mild diabetic curve. There was no spillage of sugar in the urine.

(4) Serum proteins: Total 6.3 (normal 6.7), albumin 3.8, globulin 2.5, ratio A/G—1.5.

(5) Cephalin Flocculation = ±/ 2+ (24 /48 hrs.) within normal variation.

(6) Bromsulphalein — Liver Function Test (5 mgm. per kilo body weight) 6/0; (30/60 min.) within normal range.

Duodenal Studies: (1) Bile—"A"—"B"—"C".

(a) Normal concentration. Negative for pus, crystals and parasites.

(b) Culture—"A"—Yeast cells on S. S. media "B"—Yeast cells on S. S. media. Gram positive cocci in pairs and chains, identified as a non-hemolytic streptococcus.

(2) Enzymatic

(a) Tryptic activity 33.3 units (normal 25-50)

(b) Lypolytic activity 0.8 units—(normal 0.2-2.0)

(c) Amylolytic activity 1.2-1-4 units—(normal 2.5-5.0)

Roentgenograms

1. Barium enema—colon negative for pathology.
2. G. I. series—stomach and small intestine negative for pathology.
3. Flat plate—numerous small, discrete, dense shadows probably calcifications are noted throughout the whole of the pancreas.
4. Cholecystogram—gallbladder normal in size, contour and position; no calculi.
5. Chest — lung fields are clear. No evidence of metastasis. A large and small metal foreign body is present. Healed fracture of a rib. No calcifications of the aorta.
6. Osseous structure—skull, long bones, hands and feet; no cystic changes are demonstrated.

The patient remained with us for six to eight weeks. Rest and diet were the only treatment we could offer. With this, he gained four pounds. His diarrhea slowed down to two to four movements daily. He never complained of abdominal pain.

Because of the nature of his illness, he was returned to the States. I have been unable to trace him and, reluctantly, cannot offer any follow-up.

With my "bag" full of data, I returned to this country in late 1945 with intentions of furthering my knowledge of calcareous diseases of the pancreas. The results of this are now offered to you.

HISTORY

As far back as 1667, De Graff described pancreatic lithiasis. Allen in 1903 first reported a case of calcification of the pancreas. It was Beling in 1940 who called our

attention to the relationship and yet the difference between the two conditions.[10]

INCIDENCE

Until 1938, over a period of 271 years, only 204 cases were reported.[12] In the subsequent five years, the total ran to 232 of which 19 were of the disseminated variety.[6]

Calcareous disease of the pancreas is obviously then an uncommon condition. The diagnosis in the past was usually made at autopsy and occasionally at the operating table. The increased use of the roentgenograms had made its recognition more common. Autopsies in three different hospitals revealed a total of 26 cases in over 70,000 consecutive postmortems[11] (0.04 per cent). In routine roentgen examinations of the abdomen coupled with the gastrointestinal series, calcifications were demonstrated in 0.07 to 0.13 per cent.[2]

This disease is found predominantly in the male. The average age is 40 years, although one case has been reported with the onset at the age of six.

ETIOLOGY

The etiology is not definitely understood. There seems to be no doubt about the fact that pancreatic lithiasis and general pancreatic calcifications are independent pathologic entities, which, however, probably have a common etiologic basis.[13] It is believed that either an inflammatory, infectious or necrotic process in the pancreas is responsible for the alteration of the chemical composition of the pancreatic secretions and the subsequent deposition of calcium in the ducts[4] and parenchyma. The contributing factors seem to be: (1) bile tract and gallbladder disease; (2) squamous metaplasia of the ducts; (3) desquamated, cornified epithelium may serve as a nidus for calcification in the ducts[8]; (4) invasion by the blood stream or lymphatics of infection from adjacent structures; (5) mumps[7]; (6) chronic alcoholism has been found so consistently, that it is considered more than coincidental (45 per cent of cases).[3]

PATHOLOGY

The exact manner and time necessary for the formation of the calcareous deposits are again unknown. Recent work on acute pancreatic necrosis[9] offers a plausible solution. These observers found large quantities of calcium in acute pancreatic lesions and at the same time noted a moderate fall in the serum calcium of these patients, especially between the third and eleventh day. They reported three cases of tetany associated with acute pancreatic necrosis. The theory[8] is proposed that there is a splitting of the neutral fat by the lipase of the pancreatic juices resulting in glycerin, which is absorbed, and fatty acids which combine with the calcium of the blood to form insoluble soaps.

The calcareous depositions are variously described as organic, containing 12.7 per cent lime salts and inorganic with 93 per cent calcium carbonate.[2]

True stones are present in the ducts and ductules, usually multiple, grayish or brownish gray, hard, rough and angular. False stones, that is, parenchymal concretions, are chalky, gravel-like, in large or small collections, and frequently are mixed with yellowish or chocolate colored organic matter.[8]

The consequences of calculi may be serious. The obstruction of the pancreatic duct, if complete, may lead to an acute pancreatic process, but if incomplete the changes are of a different type. Atrophy and progressive fibrosis with more or less destruction of the parenchyma results. The islets of Langerhans are not significantly involved until the disease is far advanced.

SYMPTOMS

In 1920 Pratt maintained that there was no clear cut symptom complex for pancreatic lithiasis. With the passing of 25 years, this statement is still true, even in the most advanced lesions.

The location, size and mobility of the calculi have much to do in molding the clinical picture. Pain, diarrhea, and symptoms of diabetes mellitus are the most common complaints.

Pain may be dull and transitory or severe and colicky. In some it may resemble pylorospasm or gastrospasm of such a nature as to appear to be functional or psy-

chogenic.[4] The colicky type usually cannot be differentiated from biliary colic, but the radiation to the left side of the abdomen and into the left costovertebral angle is highly suggestive of its pancreatic origin.

In about half of the cases there are signs of pancreatic insufficiency, manifested by frequent, bulky, pale stools containing large amounts of fat and numerous undigested meat fibers. This steatorrhea and creatorrhea are not constant even in this 50 per cent.[4] The marked asthenia and inanition associated with advanced insufficiencies are worthy of mention.

The findings of glycosuria and hyperglycemia as evidence of diabetes mellitus are noted in only about 45 to 50 per cent of the cases of calcareous diseases of the pancreas.

Jaundice is not an infrequent symptom. It was found in 27 per cent of the cases subjected to surgery by Haggard and Kirtley. This is probably due to an obstructive phenomenon, although there may be some few resulting from secondary hepato-cellular damage.

PHYSICAL EXAMINATION

There are no physical findings considered to be pathognomonic. Epigastric tenderness associated with varying degrees of guarding may be present. Icterus and hepatomegaly have been observed.

LABORATORY FINDINGS

The use of the pancreatic function tests have not reached the stage of general availability which would make them particularly helpful in the diagnosis. In acute episodes, an elevation of the blood amylase and lipase[14] may be of definite aid. However, in the chronic phases, these tests are of no value.

The urinalysis reveals a positive sugar test in 20 to 34 per cent of the cases. An increase in the urinary amylase, corresponding to the increase of the enzyme in the blood, is found occasionally during episodes of acute pain.

The glucose tolerance test will denote the degree of hyperglycemia and the severity of the diabetes.

Fecal analysis yields in some cases undigested fats and meat fibers.

The study of the duodenal enzymes may show a normal, decrease or complete absence of pancreatic juices.

ROENTGENOLOGIC EXAMINATION

The most dependable sign, characteristic and diagnostic of calcareous disease is found in the roentgenogram. The shadows are usually dense, multiple and grouped and, in the P-A view, may be seen on either or both sides of the vertebral column, corresponding roughly to the axis of the pancreas. Frequently, they are best visualized in an oblique view.

DIAGNOSIS

The diagnosis of most of the cases has been made accidentally during routine roentgenograms. The symptoms of epigastric pain, pancreatic insufficiency and diabetes should make one highly suspicious of a pancreatitis, but it remains for the x-ray department to demonstrate conclusively the calcium deposition.

In the acute colicky phase, the differential diagnosis should include biliary colic, perforated viscus, renal colic, coronary thrombosis, mesenteric thrombosis, intestinal obstruction and tabetic crisis.[14]

When diarrhea is the presenting complaint, one must rule out the various types of chronic colitis, sprue and idiopathic steatorrhea.

TREATMENT

In asymptomatic pancreatic calcification, therapy is unnecessary. However, regular, interval examinations should be done in order to reveal early evidence of pancreatic failure.

Surgery is usually advised to those who have colic. In capable hands, there is relatively little difficulty caused by postoperative pancreatic fistula or by a reactivation of the pre-existing pancreatitis.[4] In the acute attacks, opiates and fluids are given freely. Ephedrine has been advocated to reduce the volume of pancreatic juice.

If the pancreatic damage is extensive, palliative therapy is the only recourse. The treatment proposed by Beazell, Schmidt and Ivy consists of the administration of enteric coated pancreatic tablets, 8 grams with each meal, 4 before and 4 after.[14] This is increased or decreased, depending on the

therapeutic response. Bile salts, vitamin B complex as well as the fat soluble vitamins may help. If there is an associated achylia gastrica, dilute hydrochloric acid may reduce the number of bowel movements and aid in digestion. The permanent limitation of fat is the only dietary restriction.[14] When diabetes mellitus enters the picture, insulin is used accordingly. Patients with fatty changes in the liver may be benefited by use of lipocaic.

COMPLICATIONS

The complications of calcareous disease may include the following:

1. Pancreatic — cysts, pseudocysts, abscesses and carcinoma.
2. Liver — fatty changes, hemachromatosis and portal cirrhosis.
3. Stones may perforate into an adjacent organ or peritoneal cavity with secondary hemorrhage.[15]
4. Predisposition to pulmonary disease as tuberculosis, suppuration and gangrene.[8]

COMMENT

For all practical purposes, you have the story of calcareous diseases of the pancreas. However, before closing, I should like to point out a few facts relative to this case report.

The credit for the diagnosis again belongs to the roentgenologist.

It was intensely interesting to study the disturbed physiology in such an extensively damaged organ. On examining the roentgenogram, I immediately wondered just how much function remained. As you have already seen, the tryptic and lipolytic activities were normal. This is consistent with the literature. The question then must arise, why the steatorrhea and creatorrhea? One observer has contended that this is the result of hypermotility of the bowel secondary to a vitamin deficiency state.[1] The roentgenologic evidence of such a change in the small bowel pattern is lacking in this case. The answer must be then not in the quality of the pancreatic juices but in the quantity secreted. This is brought out more emphatically by the fact that the patient was able to digest the Schmidt diet, but was unable to handle the more liberal regular ward food.

The positive culture of non-hemolytic streptococcus in the gallbladder will find much favorable comment among those who designate the bile tract as the original focus for this pancreatic disease. On the other hand, the cholecystogram and microscopic study of the non-surgical biliary drainage reveal no other evidence of pathology and hence lend no support to this view.

The serum proteins, cephalin flocculation and bromsulphalein, would indicate that the liver had escaped injury.

There seems to be ample proof that there is no parathyroid or chronic calcium metabolic disturbance associated with calcareous disease of the pancreas.

The latent diabetes in this case is worth while mentioning, only to say that there is apparently no correlation between the degree of diabetes and the involvement of the pancreas.[4] This is not at all surprising in view of our present knowledge of diabetes.

That this disease is rare[11] remains to be challenged only by those[4] who have found the incidence higher in their experience. However, regardless of our individual feelings in the matter, we all would agree with Korte who said "that the diagnosis of the disease of the pancreas would be far commoner if the physician would remember that his patient had a pancreas."[4]

I gratefully acknowledge the assistance of Dr. Sam Levi of the x-ray department and Dr. I. I. Zipkin of the biochemistry department.

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DISCUSSION

Question—What are the chances of detecting this condition in routine x-ray examination of the gastrointestinal tract

Answer—In answering this question, I should like to call your attention to slide 3. Despite the fact that calcifications of the pancreas are more dense roentgenologically than the bones of the finger and bladder calculi, they are hardly distinguishable from the barium in the gastrointestinal tract. As you see, it would be a very difficult job, even in this extensive calcification, to interpret the densities above the stomach. Unless one was exceedingly sharp, he would pass those up as part of the small bowel pattern. Certainly, when only one stone or a small patch of calcification was present, it most probably would be overlooked. Beling and his co-workers[11] have advocated a flat plate of the abdomen to precede all gastrointestinal roentgenograms whenever calcareous disease of the pancreas is suspected.

Question—Was there a history of previous attacks suggestive of acute pancreatitis?

Answer—Yes, I would say so. The history of the original attack, which required morphine for relief, certainly had every appearance of acute pancreatitis.

Question—Have you any plausible explanation why there should be this type of calcium deposit in the pancreas when equally as severe pathology, or cell damage elsewhere does not result in this type of calcium deposit?

Answer—I do not think that this type of calcium deposition is peculiar to the pancreas. We see large calcifications in other organs, such as blood vessels, muscles and tendons and lymph glands. Why certain tissues respond with calcium deposition and others with fibrosis, I am unable to say. The presence of the fatty acids and formation of calcium soaps may be the answer, but I will leave that phase to the pathologists.

Question—What is the relation of bile tract disturbances to this condition?

Answer—It is interesting in reading through the literature to find that except for calcareous disease of the pancreas, all other pancreatic inflammations and infections have been associated with or are secondary to infections of the bile tract. Calcifications of the pancreas are found predominantly in the male, whereas biliary disease is most common in the female. Only in 30 to 45 per cent of the cases reported, has there been any evidence of biliary disease.

THE FENESTRATION OPERATION FOR OTOSCLEROSIS:*

ITS POSSIBILITIES AND ITS LIMITATIONS

J. V. McLAURIN, M. D.

BATON ROUGE

In the whole range of medicine there are few conditions more absolutely discouraging from the standpoint of curative therapeutics than deafness. It is not a lethal condition, it is true, nor does it physically incapacitate the victim. However, it does disable him, partially or entirely, in his social and economic relationships, and the psychologic effect is profound, more profound, many observers state, than even the reaction to blindness.

THE ANATOMICO-PATHOLOGIC BASIS OF LOSS OF HEARING

Before discussing the therapy of loss of hearing, let me remind you of the anatomy and physiology of the ear. To state it as simply as possible, air-borne sound waves enter through the outer ear and strike against the tympanic membrane. The vibration thus set up in this membrane is communicated to the ossicles, the chain of small bones in the middle ear. The last of this chain, the stapes, plugs the oval window opening into the inner ear. The vibration of the stapes sets in motion the fluid in the inner ear, and sound impulses are thus transmitted to the organ of Corti, from the organ of Corti to the auditory nerve, and from the auditory nerve to the brain for interpretation. The interruption of sound impulses at any point in the chain of transmission will obviously produce impairment of hearing.

Acquired deafness is divided into two categories, conductive deafness and perceptive deafness. Conductive deafness arises from a pathologic process in the middle ear or the external ear which prevents sound waves from reaching the cochlea. Perceptive deafness arises from a pathologic process in the cochlea itself, or in the nerve pathway. In numerous instances the pro-

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cess is dual, though changes in the outer or middle ear or in the auditory nerve may predominate.

THE PRESENT STATUS OF THERAPY

With the possible exception of the vitamin therapy recently practiced for certain cases of perceptive deafness, it can be stated categorically that no medical treatment ever devised has permanently improved the hearing of a patient whose impairment was on an organic or structural basis. If the hearing is actually or permanently improved after such therapy it can be assumed that the disability was of psychologic origin. In carefully selected cases of this kind, appropriate treatment, particularly the new method of narcosynthesis used with such success in certain Army hospitals, has produced excellent results.

It is well to point out that while the whole Army Program for the Aural Rehabilitation of the Deafened and Hard of Hearing has been an outstanding success, it is literally a rehabilitation program. It implies an acceptance of the *status quo*. The impairment of hearing is admitted and the patient is taught to adjust himself to it; his degree of auditory impairment is studied by scientific methods. He is fitted —very carefully indeed—with the hearing aid best suited to his special difficulty, and he is trained to use it. He is taught lip reading. He is taught to use to the best advantage such hearing as remains to him. He is given vocational guidance and occupational training as necessary. By these and other measures he learns to live with his infirmity and to rise above it, but his hearing, *per se*, does not improve.

In the light of present knowledge, as has been already mentioned, very little indeed can be done for deafness of nerve origin. Within the past decade, however, a great deal has been done by surgical measures for the variety of hearing loss originating in a middle ear disease known as otosclerosis, though that statement, as I shall repeatedly emphasize, justifies no generalizations as to either universality of treatment or uniformity of results.

OTOSCLEROSIS

Otosclerosis, a term applied both to the lesion which causes a special form of deafness and to the resulting deafness itself, is still of unknown etiology. Infection, abnormalities of development, and defects in the vasomotor mechanism are among the most important theories suggested to explain it.[1] There is some degree of logic in each suggestion, but proof for any single theory is not yet conclusive.

Otosclerosis may be defined as an overgrowth of spongy, vascular bone within the labyrinthine capsule. Its mere presence does not imply loss of hearing. It has been found repeatedly on postmortem examination in persons whose hearing was never affected. The location and extension of the process determines whether or not it is associated with loss of hearing. It may occur in any area of the otic capsule, but by far the most common site (so common, in fact, that it may be called the site of predilection) is the inner wall of the middle ear, just anterior to the oval window. From this point of origin the bony process easily extends to involve the stapes and lock its footplate. As it causes first diminution of mobility and later immobilization of the stapes, increasing impairment of hearing is the inevitable result, because sound waves cannot reach the inner ear. The degree of bony pathology, however, and the degree of auditory impairment are frequently not correlated.

Clinical otosclerosis tends to become manifest late in adolescence or early in adult life. It is much more frequent in women than in men, and many observers believe that it is likely to become more marked with successive pregnancies. Its most striking clinical characteristics are its insidious onset and its slow but steady progression. As the patient grows older, it is frequently associated with nerve degeneration, which in late cases may overshadow the otosclerotic process.

THE FENESTRATION OPERATION

Historical Note: To understand the development of the fenestration operation for otosclerosis, which has been described[2] as "undoubtedly the greatest otologic contri-

bution to medical science of the present generation," a brief historical note is necessary.[3] Since the region of the oval window is the site of predilection for otosclerotic changes, it was natural that the attention of otologists seeking a means of relief for deafness originating in these changes should have been focused upon that area. The first surgical procedures, performed late in the eighteenth century, consisted of a simple opening into the mastoid process. In 1876 Kessel created a fistula into the middle ear by extraction of the immobilized stapes. In 1897 Passow opened the promontory near the oval window, and occasional surgeons over the next two decades created fistulas in various other locations. In every instance immediate improvement in hearing seems to have resulted as soon as the fistula was created, but in no instance was the improvement more than transient. Sometimes regression from the previous level of hearing followed the brief period of improvement, and the operations, aside from their unsatisfactory results, were looked upon with disfavor because of their obvious dangers to life.

After 1917 the attack upon the problem became more intensive. In that year Holmgren made a fistula on the upper curve of the anterior vertical canal and at almost the same time Barany made a fistula into the horizontal canal and covered the opening with a pad of fat. In the latter case improvement in hearing lasted for 14 days. In 1920 Holmgren, using magnifying optical instruments, created an opening into the promontory and covered it with mucoperiosteum, again securing transient improvement in hearing. Two years later he utilized the same technic on the horizontal semicircular canal, and while improvement was not very marked in any instance, it was occasionally permanent.

Shortly afterward Sourdille devised a complicated three-stage operation, by means of which he was able to keep the new window in the horizontal semicircular canal open longer than had been previously possible, and Holmgren, encouraged by these results, continued to refine his own technic. By 1936 he was able to report 34 patients operated on with no deaths and no serious complications; improvement in hearing was constant, and in many instances it was marked.

Holmgren's operation was taken up by Lempert,[4, 5] whose original technic has been successively modified until it now consists of two basic steps: (1) the creation of a fenestra into the surgical dome of the external semicircular canal, and (2) the immediate obliteration of the newly created fenestra with a closely fitting but *mobile* stopple fashioned from auricular cartilage, the same material which covers the inner surface of the normally functioning stapedial foot. The operation thus reproduces as far as is mechanically possible the anatomic and physiologic background against which normal hearing is achieved.

A number of important considerations concerning the operation should be emphasized. The new window is located in the surgical dome of the vestibule, which is adjacent to the area in which the natural oval window is located but which is not the site of predilection for otosclerotic changes and in which, so far as the records show, they have never been found. The insertion of the new stopple not only recreates the normal condition of a mobile stapes but also greatly reduces the risk of labyrinthitis. This can never be disregarded in this vital area. The use of dental drills for much of the bone work and the use of optical instruments to provide magnified vision for the surgeon permit delicacy of technic and somewhat reduce the difficulties of the intricate procedure. The use of constant irrigation during the operation assures the removal of all bone particles which, as has been shown experimentally, may serve as foci for new bone growth.

What the fenestration operation amounts to is a by-passing of the bony growth in the natural oval window and the creation of a new window in an adjacent area not likely to be the site of a similar bony change. It is no more curative than is a gastro-enterostomy for an obstructive gastric ulcer, but it is quite as useful and con-

siderably more logical, since under no circumstances could the offending aural process be extirpated. Lempert,[5] whose experience with the fenestration operation is the most extensive in the world, believes that he has accumulated sufficient evidence to advance the theory that stapedial fixation, if left unchecked, eventually causes atrophy and degeneration of the auditory nerve, which becomes irreversible if it is not interrupted. If this theory is correct, the fenestration operation is quite as logical as cataract extraction done before degeneration of the optic nerve occurs. Whether the operation is done early or late, however, the fenestration operation has absolutely no immediate direct effect upon auditory nerve function.

Selection of Cases: The most important consideration in the fenestration operation is the proper selection of cases. The classical picture of the patient with otosclerosis who is likely to be helped by the operation is about as follows:[5]

1. There is a family history of hereditary deafness, and loss of hearing may become manifest quite early in life.

2. Deafness for air-borne sound is slowly or rapidly progressive in degree and extent. It begins first in the lower frequencies but may ultimately involve the higher frequencies. It is usually bilateral, though it does not necessarily appear at the same time in both ears. The patient frequently states that hearing loss is much less marked in a noisy than in a quiet environment.

3. Unilateral or bilateral tinnitus is frequently present and may be mild or severe and continuous or intermittent.

4. Examination often reveals atrophy or extreme translucence of the tympanic membranes, through which the anatomic structures of the middle ear are clearly visible. The mucous membrane lining the promontory of the cochlea is frequently congested and pinkish. The Eustachian tubes are patent.

5. Audiometric testing demonstrates that inflation of the Eustachian tube does not improve the hearing temporarily, though on clinical testing the patient will frequently claim that it does.

6. Audiometric testing also reveals:

(a) Superiority of hearing for bone-conducted over air-conducted sound, particularly for low frequencies.

(b) Maintenance of hearing for bone-conducted sound, though hearing for air-conducted sound may have sunk to a low decibel level.

7. The Weber test is lateralized to the worse ear if the hearing for bone-conducted sound is better in that ear. However, if hearing for bone-conducted sound is equal in both ears it is not lateralized.

Since in otosclerosis as in most other medical conditions the classical picture is the exception rather than the rule, it is practical to select patients for the fenestration operation on the following basis:

1. The patient's hearing for normally spoken conversation has dropped to a level at which it is no longer useful or serviceable or practical for everyday social and economic contacts.

2. The tympanic membrane is intact, and there is no evidence of middle ear inflammation.

3. Audiometric testing reveals:

(a) That hearing for air-borne sound has dropped below 30 decibels in the conversational frequencies.

(b) Hearing for bone-conducted sound is prolonged over and above hearing by air conduction in all the frequencies in which hearing by air conduction is impaired.

In other words, the fenestration operation for otosclerosis is, to put it very bluntly, a good gamble when impairment of hearing in the conversational frequencies can be demonstrated not to be the result of degeneration of the auditory nerve and when there is no evidence of gross pathology in the tympanum or the middle ear. As a general rule it should be limited to adolescents and young adults, since degeneration of the auditory nerve becomes more and more likely as age advances, and hearing by bone conduction should not drop more than 20 or 25 decibels at any point

in the conversational range. It may be, as Lempert[5] suggests, that these indications may be further liberalized in the future, but for the present it would be well for the otolaryngologist with a less wide experience than his to observe them rather carefully, though it is equally important that he should not permit his own faintness of heart to deny the operation to patients whom it might help.

Results: To date, only a limited number of otolaryngologists have performed the fenestration operation often enough to permit statements of results. Lempert,[5] who has done well over a thousand cases, reported in 1945 that of 815 patients so selected that improvement could reasonably have been expected, 571 had been completely rehabilitated and 92 partially rehabilitated; 118 were not improved, and 34 were worse than before operation. Of 309 patients operated on between three and seven years before the date of the report, 187 had been restored to practical, serviceable hearing which had been continuously maintained, while in 37 others improvement was achieved and maintained but did not reach practical levels; 65 showed no improvement and 20 had become worse. Day[2] in 1945 reported 75 cases, in 54 of which operation had been done between six months and three and a half years before the date of the report; 43 patients, 80 per cent, had been restored to practical hearing levels, including three in whom revision of the original fenestra had been necessary because it had closed.

Standards of postoperative evaluation are, of course, extremely important. Lempert[5] regards a cure as complete if improvement in hearing has lasted for at least a year, though as a matter of fact it is rather generally agreed that if improvement is maintained for six months, the chances are that it will continue. Improvement must be of a degree to permit complete rehabilitation for social and economic contacts as a result of restored hearing, which must be improved to approximately the 30-decibel level or better for the conversational frequencies 512, 1024 and 2048. He regards a patient as partially rehabilitated if his hearing is improved over what it was before operation but not to the practical 30-decibel level in the frequencies mentioned. While testing of postoperative hearing must be done by audiometer, and is preferably done by a dispassionate observer, without other contact with the patient, Day's[2] criterion is useful: If hearing improvement does not reach the level for the comprehension of normal conversation, the operation is a failure, and the best that can be said for the result is that the patient's hearing is not as bad as it was before.

SCOPE AND EVALUATION OF THE FENESTRATION OPERATION

Certain facts about the fenestration operation should be made very clear. In the first place, it is a tedious, time-consuming, delicate, difficult procedure. It can be learned only by exacting application and it should not be undertaken by otolaryngologists who will not take the time and effort thus to learn it. As Lempert[4] puts it, every step leads to the next step, and if a single step is done carelessly, the operation is doomed to failure.

In the second place, the operation is by no means free from risk. It is at least as dangerous as any operation done in the mastoid area, and perhaps more dangerous than most, though in the hands of a competent surgeon this risk is reduced to the irreducible minimum. Careful aseptic technic, both at operation and in the postoperative period, has almost eliminated the risk of infection, but it occasionally occurs unexpectedly, just as it does after any other surgical procedure, even though it is done in a clean field, and the resulting damage to the organ of Corti may be disastrous. No surgical procedure, of course, is free from risk, and the endolymphatic labyrinth and the organ of Corti have been described as the most delicate anatomic structures in the human body. Finally, as Lempert[5] points out, the performance of the fenestration operation does not make the patient immune to untoward non-aural accidents during it; he himself lost two patients from coronary thrombosis soon after operation.

Complications in the hands of a competent surgeon are not the rule. The facial nerve, since it is directly visible, is not likely to be injured except by some accident, though a transient facial paralysis is frequent and may last as long as three weeks. Vertigo after operation may be very severe, though it usually clears up almost entirely by the time the patient has left the hospital, that is, within eight to 14 days after operation.

I am among those who believe that the fenestration operation represents the greatest advance in the treatment of deafness which has ever been made, but I have been deliberately unenthusiastic in my presentation of it. I think it extremely important to emphasize its limitations, its dangers and its difficulties if only because of what it has suffered at the hands of some of its advocates. Nothing more unfortunate could possibly have occurred than the publication in the February, 1945, *Readers' Digest*[6] of an article on the operation which had first appeared in *Hygeia* and so had, at least for lay persons, the semi-official blessing of the American Medical Association.

The title of the article, "And The Deaf Shall Hear," sounds well but is completely erroneous. To be accurate, it should have read, "A certain proportion of a certain category of deafened persons have some chance of regaining some hearing." The statements in the course of the article deserve to be characterized as the author herself characterized the first description of the fenestration technic, that they sound "almost too good to be true," which is precisely what they are.

Whether, as the article states, the greater proportion of the estimated 15,000,000 deafened persons in the country have otosclerosis is most decidedly open to doubt. That 98 per cent of the deafness caused by otosclerosis can be cured by the fenestration operation if the nerve is intact is certainly not true. Some observers put the proportion at 10 per cent, though I myself believe that that is too low. That the operation is either painless or free from risk is equally untrue.

Finally, those who are familiar with the possibilities of the fenestration operation as well as with its limitations can only deprecate such facile statements as "Just as cataracts no longer mean sightless eyes (a statement that can be described only as verging on the reckless) so otosclerosis ceases to condemn its victims to a world of silence," and "For the first time in medical history the doctor no longer needs to inform the deaf patient that there is no cure, no hope, but lip reading or a hearing aid." Quite a number of deafened persons will still do much better with lip reading and a hearing aid than with the fenestration operation, and an unfortunately large number of patients with otosclerosis will still continue to live in a world of silence.

Such glowing accounts of the operation do no good. They raise false hopes, which the otologist must dash when he refuses to operate in inappropriate cases and leaves the patient thinking that he has been denied his chance of cure. The operation itself is discredited if the pleadings of the patient lead the surgeon to operate against his better judgment. Patients who see such failures will not apply for aid, even though they might be in the group which could be helped.

The best interests of the deafened patient demand that his hearing impairment be investigated dispassionately, clinically and audiometrically; that it be determined whether his hearing impairment is: (1) of the otosclerotic type, and if it is, (2) that it is within the range likely to be helped by surgery; that he then be told, beyond chance of misunderstanding, the possibilities of relief and the discomforts and risks of the operation, though it may fairly be pointed out that even if his hearing becomes unilaterally worse after operation, that consideration is little more than academic, since it is already not serviceable. In the light of these facts the patient may then be permitted to decide for himself whether or not he wishes to accept the chance of relief which the fenestration operation offers him.

SUMMARY

The development of the fenestration operation has been traced in its essential principles and it has been pointed out that it is applicable only to the otosclerotic variety of deafness and only to a limited number of carefully selected cases within that category. It is emphasized that unwarranted claims of good results and the performance of the operation in inappropriate cases will discredit what has been correctly described as the most important advance in the therapy of deafness which has ever been made.

Note: Since this paper was completed there has come to my attention a report by J. A. Sullivan (Canad. M. A. J., 53:543, 1945) on 410 fenestration operations, with complete social and economic rehabilitation in 246 patients or 60 per cent. The writer emphasized the importance of exactitude in each step of the procedure.

Also published since this paper was written was a report by Shambaugh (J. A. M. A., 130:999, 1946) on 930 fenestration operations. The highest percentage of improvement, maintained for two years or more, was achieved with the nov-ovalis operation; 246 of the 337 patients or 73 per cent who were operated on sufficiently long ago to be included in the follow-up and who returned for audiographic study showed permanent improvement. The paper also includes an extensive experimental study.

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DISCUSSION

Dr. W. A. Wagner (New Orleans): Certainly we have enjoyed this most interesting and timely subject, and certainly the essayist has brought to us a very good introduction of this new operation, fenestration for otosclerosis. I would like to discuss the subject from the standpoint of practical experience. After six and a half years or more of practical experience with this procedure, it strikes me that there are some fundamental conclusions that one should draw from the end results. There is no question of doubt but that the operation has contributed a big advancement in the handling of that specific type of deafness. There is no question of doubt too, that the operation has some fallacies. For example, I have found out from my personal experience that the operation does restore hearing in otosclerotic deafness. It perhaps can restore as much as 35 decibels, in my experience. It has given excellent rehabilitation. Now, granting that fact, the question then is debatable as to whether a patient should subject himself to a fenestration operation or to a hearing aid. I will admit that there are many handicaps in the use of hearing aids which are perhaps gotten rid of with a successful rehabilitation by means of operation, but let us look at the fallacies of operation. Granting that I can restore 35 decibles after the operation, I can not and have not, been able in the past to maintain that 35 decibel improvement. As most of you know, the window will close either by osteogenesis, fibrosis or irritated labyrinthitis as result of procedure.

There are several procedures that have been described to us; the first operation, of the horizontal canal. I went through that. I opened the horizontal canal and my cases closed.

Then the nov-ovalis operation, whereby the window is made at the junction of the ampulla of anterior vertical and horizontal canals or what is known as the surgical dome of the vesicle. That has been the most successful procedure to me, to date.

Another more recent procedure whereby the cartilage of the helix is placed as a stopple in the newly created window. My experience in the stopple operation is more discouraging that results obtained from opening the horizontal canal and I returned to the nov-ovalis procedure.

I think Dr. Shambaugh in Chicago has contributed quite a lot to the technic in maintaining an open window. For example, he uses a procedure he calls the continuous flow or irrigation procedure. In making his window, continues the flow of saline at body temperature over the operative field. I have found, in using continuous irrigation it interferes with my procedure so I use periodic irrigation. Dr. Shambaugh thinks that the continuous irrigation is superior, he may be right; I cannot contradict him there but let's get back to the end results again.

No matter whether you use the original fenestration in the horizontal canal or non-ovalis, or stopple, my experience has been that the majority of the windows close. I recently made a six year report on end results. I think this represents perhaps an unbiased report of my own experience. Out of 17 cases over six years old to date, there are three rehabilitations. Now out of that group all but two were improved, shortly after operation. All of the 11 have lost some of that improved hearing. Only four have lost a greater amount of hearing than the hearing preoperatively.

My conclusions are these: That if we expect to raise our percentage of rehabilitations we must more carefully select our cases. I have found out that in this group and the recent groups final restoration of hearing may be considered after one

year—and one year is considered a sufficient time to expect the hearing to be maintained without closure of the window. In those cases whose hearing gain ended over the one year period about 15 debicels was maintained. Now let me reiterate that and qualify. The maintained improvement has been on an average of about 15 decibels, therefore we should try, in my experience—perhaps I should change too—I should attempt to select my cases in the group whose hearing losses are greater than 30 decibels and not to exceed 50 decibels. If I could select all cases up to 50 decibels I think I could show a larger percentage of rehabilitation. In those individuals whose hearing loss exceeds 60 decibels I finally admit I do not believe I can ever rehabilitate those individuals by the present technic of the fenestration operation. I sincerely mean that, unless my technic is changed.

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DISEASE AND THE AGED*

W. A. SODEMAN, M.D.†

NEW ORLEANS

The era of diseases of the aged is not starting. It has already begun! In Roman times life expectancy at birth was about 23 years. In 1900 in the United States the figure had risen to 47 years, an increase of 24 years in nearly as many centuries. In 1940 it had risen to 63 years, a rise of a similar period in only four decades. At present over 80 per cent of newborns will live to 50 years of age, over 60 per cent to 65 years. There are now in the United States about 9,000,000 persons who have attained the age of 35. With present trends this figure will be 22,000,000 by 1980. I am not going to burden you further with statistics which have been presented to you many times before. Suffice it to say we are fast becoming a nation of oldsters.

Much of this change has resulted from the application of preventive measures not only to control the environment but also to help the development of increased resistance of individuals to infectious disease. Here, public health procedures have been important. When to these measures are added the use of newer chemotherapeutic and antibiotic agents in treatment, a further reduction in mortality from infections and a rise in the figures for death from degenerative disease can be visualized in the next few decades. The lead gained by diseases of older age groups in our mortality figures in the past forty years, as shown in table 1, is likely to be increased more rapidly than present estimates indicate, because of recent astounding advances in the treatment of infections. Two groups of disease alone, cancer and cardiovascular disease, will probably increase to such an extent that they alone will appreciably affect the total mortality rate. Add to this diabetes mellitus and the rest of the diseases and we can see that disease and the aged is an ever expanding problem which we, the medical profession, cannot meet with the lethargy and neglect which has characterized our approach in the past. A constructive active program is necessary.

TABLE 1

Rank of Leading Causes of Death

1900	*1940*
1. Tuberculosis, all forms.	1. Diseases of the heart.
2. Pneumonia (probably including influenza).	2. Cancer, all forms.
3. Diseases of the heart.	3. Accidents.
4. Chronic nephritis.	4. Cerebral hemorrhage.
5. Accidents, all forms.	5. Tuberculosis, all forms.
6. Cerebral hemorrhage and paralysis.	6. Nephritis.
7. Cancer, all forms.	7. Pneumonia.
8. Diphtheria.	8. Diabetes.
9. Diarrhea and enteritis.	9. Diseases of liver and gallbladder.
10. Typhoid fever.	10. Arteriosclerosis.

Comparison of the Ten Leading Causes of Death in 1900 and 1940. Note that three groups of infections are no longer in the list in 1940 and two others (tuberculosis and pneumonia) have lost their number one and two positions.

Beginnings have been made in the approach. In 1805 Benjamin Rush discussed the problem. Others, following him,

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sought means of prolonging the natural span of life. Such were the hopes held out for acidophilus milk and for operative procedures removing several feet of the intestine. But it was J. L. Nascher who introduced the modern concept of the care of the aged. He died in 1944. His first important article appeared in 1908[1], and his textbook entitled *Geriatrics* appeared in 1914. This present year has seen the establishment of the journal, *Geriatrics*, devoted to research and clinical study of the diseases and processes of the aged and aging.

The problems of aging fall into three categories: (1) the biology of senescence as a process; (2) the clinical problems of senescence, and (3) the socio-economic problems of aging. The first two are always important to the practice of medicine, the third may be especially so in industrial medicine. The process of aging produces many important changes. The desiccation of tissue, slowing of cell division, atrophy, diminution of muscle strength, reduced elasticity of tissues and impaired keenness of the special senses, create special problems of old age. Patients react differently to disease. Therefore, their symptoms may be different. The older body differs so the signs may be different. Changed chemistry may influence laboratory data. Finally, treatment must fit this changed old body. Just as children are not small adults and create special problems for the pediatrician, so the elderly patient differs from the young adult and creates special problems.

Such remarks naturally lead to consideration of geriatrics as a specialty. In my opinion there is little general need for specialization in geriatrics. The term, like the term *psychosomatic*, is important in that it focuses attention on a problem. To be sure, in some large centers and densely populated areas, some may devote their full time to the study and care of the aged, but by and large this must be the problem of general practitioners. It is essential to remember that many of the factors contributing to disease in old age have their beginnings in other years and treatment at that time is most important. What, then, are the problems that confront us and how can they be met?*

The measures applied in the control of the environment, such as those effective against typhoid fever, as well as those effective against such problems as dusts, fumes, and noxious gases in industry, have been eminently successful as exemplified by the changes in mortality statistics already mentioned. Such methods have been applied almost without thought given them by the individual and even the physician in large part. In urban areas good water supply and adequate sewage disposal are accepted by most of us without much thought. The effectiveness of preventive measures in suppressing infectious diseases in the past has rested upon the mass approach made possible through control of a known etiologic agent which should be blocked in the environment. In the diseases affecting the aged, such conditions do not prevail. In malignant disease, metabolic disorders, hypertension and arteriosclerotic damage, to cite a few examples, the etiology is obscure and, to our knowledge, chiefly endogenous. The clinical picture is often insidious in its onset and the course chronic and progressive.[2] Against such diseases, therefore, the preventive measures applicable to infections are ineffective. Preventive procedures, just as therapeutic measures, must be individualized. The subjects cannot be approached in mass but must be examined and studied singly. Prevention must be carried out within the limits of the practice of medicine. Some preventive measures have long been applied to the individual (typhoid vaccine, diphtheria toxoid, and so on), but in large part active preventive programs with individualized approach have been applied extensively in two fields, pediatric and obstetrical practice. As Stieglitz[2] notes, modern pediatric practice has demonstrated convincingly that the health of well babies can be improved, that guidance in nutrition,

*Geriatric medicine is a part of the larger problem of chronic disease, a field in which facilities for care are woefully lacking, except in tuberculosis and psychiatry. The broad aspects of the problem of chronic disease in general will not be discussed here.

regulation of exercise, instruction in sane cleanliness, and advice regarding wiser habits of living, do develop healthier children. It is absurd, he states, that such medical guidance should be applied only to infants and children.

Other factors produce difficulties in prevention and control of disease of the elderly. Because of lack of symptoms, early diagnosis is difficult. Then, too, there is a general low individual interest in prophylaxis. Most important is the lack of drama in preventive treatment. The patient cannot see the benefit as he does in a rapidly moving acute symptomatic picture. However, the attitude of the physician is our most important stumbling block now. Many of us in our investigation of the elderly patient look no further than for some assurance that immediately important changes are absent. If the lungs are clear and the heart compensated, we pat the old man on the back and assure him he is all right. A few of us—perhaps, we feel, more enlightened—may give him some advice as to how to avoid disease. Only too few of us, however, approach the ideal of seeing in the patient, especially when he has little or nothing in the way of complaints, the opportunity to apply the principle not only of prevention of disease but of improvement of health. This is not a new concept but a further application of the principle extensively applied in children for many years.

I have already pointed out the differences in symptoms and signs produced by disease in the old patient as the result of changes of senescence. These, together with the changed physiology producing them, cause too few of us to see in our older patient some of the advances of active disease. For example, in the elderly, emphysema and concomitant bronchial changes producing cough are not uncommon. The same is true of bronchiectasis and heart disease. Such changes make for difficulty in the recognition of other disease developing in the chest. Tuberculosis, which is known to be frequently unrecognized in the elderly, may advance in the chest, produce cough, loss of weight and other symptoms in the presence of a positive sputum and long be overlooked because old patients show little in the way of toxic effects. There are many elderly tuberculous patients in the United States in whom detection of the disease is not simple. If the physician assumes the cough and related symptoms are due to pulmonary and bronchial changes resulting from age, he will never help these patients and will fail to protect against infection those with whom they live.

Nutritional disease is another field in which vague nondescript symptoms are likely to be misinterpreted. Old people, especially if they live alone, are likely to eat a monotonous diet. Their desire for a variety of food sometimes diminishes. The difficulties of traveling to the store and of preparing food for a proper diet often lead to repeated use of the same foodstuffs. The amount of food eaten becomes reduced. I have seen many old people who have lived for weeks chiefly on bread and coffee. If dietary histories are not obtained, the weakness, numbness, incoordination, and lassitude due to subnutrition are very often interpreted as the result of arteriosclerosis or "old age." I have seen remarkable "cures" in such people effected by nothing more than adequate nutritional therapy.

Clearly then one of our problems in the care of the elderly patient is the physician's attitude toward the patient. The patient's symptoms should be given the same consideration and analysis accorded those of the younger group.

Another factor of importance in disease of the aged, again already mentioned, is the physician's lack of interest in diseases prevalent in later life. Syndromes on an arteriosclerotic basis, mild diabetes, hypertensive disease, are not curable. I have often heard the attitude expressed, "You can't do anything for them, why worry greatly about them." This is not true. I do not believe that the day of medical magic will arrive when these disturbances will be eliminated. Indeed, I think they will increase. I do not hope for a cure for the process of aging. I think it is a natural process and that beyond a certain point, we

cannot add years to our lives. But I do believe the statement that we can add life to our years. Well controlled insurance statistics show that patients with degenerative diseases who undergo physical examinations frequently and are given advice as to proper care and control of their activities do get along much better and that mortality statistics are improved. Much of the chronic disease present in older age groups, arthritis and hay fever and asthma, for example, is not killing and does not contribute greatly to mortality statistics. The relief that many people with these diseases obtain from proper medical care is tremendous.

A further consideration of importance, implied from remarks already made, is the attitude of the physician on matters of health improvement. The absence of positive physical findings or other evidences to establish a specific diagnosis is *not* the signal to dismiss the patient. His habits of eating and exercise, his rules of bodily care, factors of clothing, housing, and daily routine should be reviewed to find the points at which his health may be improved or where beginning deterioration may be blocked. This concept makes the difference between the modern approach to disease and the medicine of the Middle Ages.

Finally, I must stress again a point of importance in the care of the elderly. This is the recognition by the physician of certain peculiarities which age gives to disease. This is well known in pneumonia, for example, where the febrile response and the general symptoms do not find the dramatic expression they do in the younger group. Complications, too, are often different. I not uncommonly see, in the elderly patient hospitalized for cardiac and pulmonary disease, the development of disorientation, irritability, and emotional instability coming on rapidly and progressing until there is a clouding of consciousness or even coma. The patient may develop fears at night or have his symptoms only at that time. Muscular incoordination, rigidity of muscles and tremors may appear. Recovery usually follows proper supportive and symptomatic care. These symptoms rest upon cerebrovascular changes as a result of bed rest in the elderly. They do not occur in youth. They are a part of the problem of the treatment of disease in senescence.

The points discussed above show the benefits to be derived from the proper care of the elderly patient as an individual. Even though the numbers of our population above the age of fifty increase to the extent that, from numbers alone, the problem is a public health one, the mass approach applied by public health departments to the control and eradication of such diseases as typhoid fever and even tuberculosis cannot be applied here. The nature of the diseases is different. Sadly enough, the etiology of many of the disturbances is unknown. If the causes were discovered and if means of control were established, the approach would be radically different. With our present knowledge an attack on the environment would not seem a likely approach. Individual care would be necessary for each individual patient. The technics of the cancer detection and prenatal clinic would probably be necessary, a program which, with proper education of the public and the proper attitude of the physician, could be as well—likely even better—carried out in the office of the private physician. In geriatrics, as in pediatrics, adequate clinical facilities in the hands of an interested and alert clinician will give the best results in our present state of medical knowledge. Here, preventive procedures are individual clinical problems best cared for in the office of the practicing physician. Only the alertness and adequacy of the medical profession in solving this problem in the rapidly expanding field of geriatric medicine will settle the need for health department sponsorship of health examinations and community provision for such facilities. We have delayed too long in attacking this problem.

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DISCUSSION

Dr. E. L. Wenk (Cloutierville): There are at

present two geriatric journals being published, *Geriatrics*, official journal of the American Geriatric Society, and *Journal of Gerontology*, published by Charles C. Thomas of Springfield, Ohio.

I think Dr. Sodeman has made an excellent presentation. However, I would like to re-emphasize the use of the term annual health inventory and not health examination; the objective of the annual health inventory being to detect, by means of stress fuction tests, early degenerative processes which may not otherwise be discovered.

Dr. J. E. Knighton (Shreveport): I do not think I can add anything to the information that the doctor brought us; he has given a most excellent discussion of the subject. I am often reminded of a remark that I have heard my past associate, Dr. Willis, Sr., make on many occasions—that if we continue to live we are going to grow older, and that he did not know but one way to keep from getting old and that was to die young and not many people would voluntarily select that means to avoid old age.

With reference to how people should take care of themselves as they advance in years, I think that we should remember that as we grow older it is a natural course of events for us not to have resistance and resiliency as in earlier life and just to look at the problem in a common sense way. Only last evening a doctor friend of mine here in attendance at the meeting—he is 67 years old I believe he said—told me that when he walked from his office to the hospital there was a drug store about three blocks away where he had to rest because he felt pain in his chest. He wanted to know what I thought about it. I said that means that you are developing some inflammation with reference to yourself that you ought to heed. If a man cannot walk more than three blocks without developing pain in his chest I think every doctor knows that he should not abuse that warning. This is not only true in that particular thing but there are many other danger signals which we should heed as we pass along the pathway of life. A good many of my friends, even here in this meeting, have complimented me by telling me that I do not look older than I did ten years ago. I know they are just joking.

I am still reminded of what Dr. Willis said, "Keep living to grow older."

Question—At what age did the author of the text on geriatrics die?

Answer—About sixty-three or sixty-four.

Dr. W. A. Sodeman (in closing): I am glad some remarks were made about the stress tests in the care of elderly people. That was not a part of the thesis I was to develop so I could not say much about it. The remarks give me an opportunity to add something. Of course as we get older and as the reserve power in various organs in the body become diminished we see a greater importance in determining, by stress tests, the limits of our ability to adapt. Such tests are particularly important not only in that they complete the routine examinations given to individuals from time to time for comparative study but because they point out to you as well that a health examination is not merely a matter of taking a quick history on someone and doing a physical examination to elicit a murmur, or feel a liver, and so on. Health examinations are more than merely an examining procedure to be carried out; they should evaluate the individual as he fits into his environment and show how he reacts to it and how he acts if it is changed. Therefore you have to know something about the individual's habits, background, what he does about it, the extent to which he uses various functions in his body and, if possible, improve those things for him. You must know whether he has reached the limits or has come near to reaching the limits of some of the reserve powers in his body. If you are able to do so and carry out a review in that way, you have really investigated that individual. I do not think there is anything that has put health examinations in more disrepute than the forms as brought out for such procedures by the A.M.A., blanks where you check on a line one or another thing about a patient. Nothing will let you treat a patient in a factory way more than that. A health examination should evaluate the state of health and cannot carry out by that procedure.

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THE PATHOGENESIS OF POSTOPERATIVE ATELECTASIS*

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NEW ORLEANS

The word "atelectasis" is derived from two Greek words, "ateles" meaning imperfect, and "ektasis" meaning expansion. It was first used by Joerg (cited by Adams[1]) in 1832 to describe the lack of inflation of the lungs of newborn infants as seen postmortem. Since then its meaning has been extended to include clinical cases in which there is collapse of lung tissue due to an absence or marked diminution in the amount of air in the alveoli of a whole or of a portion of lung. Jackson[39, 40] described three types of atelectasis according to its distribution:

1. Lobular or partial (also referred to as patchy): The upper or middle third of one or more lobes is the most frequently affected part. There are scattered areas of atelectasis, usually with emphysematous

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areas between them. The most frequent location is the right lower lobe.

2. Lobar: One or more whole lobes are collapsed. The right lower lobe is the most frequent location. Pasteur originated the term "massive collapse of the lung" to describe this type.

3. Total: The whole of one or both lungs is collapsed.

HISTORY

In the early days of anesthesia, the local complications of surgery were so severe and were so frequently encountered that postoperative pulmonary complications were either entirely overlooked, or, when observed postmortem, they were interpreted as terminal conditions. However, with the development of asepsis and improvement in surgical technic, the incidence of local complications markedly diminished, so that the pulmonary complications then attracted attention and became recognized as very important sequelae of surgery. At first, the reason why these latter conditions developed appeared to be self-evident, for it seemed logical to infer that they were caused by the irritating effect of ether vapor upon the lungs. For a long time this action of ether was considered to be the only factor, or, at least, the most important one responsible for the development of these lesions. Of course, the skill of the anesthetist was seriously questioned, and was considered by some to be the most important factor involved. Even today there are those who put the whole blame on the anesthetist. As a result of this theory, all postoperative pulmonary complications were designated as either "ether bronchitis" or "ether pneumonia," depending upon the severity of the symptoms. Later, with the recognition of pulmonary embolism as a postoperative complication, at least part of the blame was diverted from the anesthesia to the surgical technic. Yet, the true picture was destined to remain obscure for a long time, because the condition, which is most frequently responsible for postoperative complications, although it had been recognized clinically as a post-diphtheritic complication by William Pasteur in 1886, was not even considered as a possibility in these cases until Sir James Barr[7] called attention to the occurrence of atelectasis following operation. In 1907 he reported it in a youth a few days following the removal of tuberculous glands from the right iliac fossa. Nevertheless its importance, as a pulmonary complication following abdominal operations, did not become apparent until William Pasteur[60, 61, 62] published a series of articles on the subject between 1908 and 1914. He first suspected, then recognized, that the so-called "postoperative pneumonia" was, in many cases, massive atelectasis rather than pneumonic consolidation. In 1914 Elliott and Dingley[26] corroborated Pasteur's observations and emphasized its clinical importance. They also indicated that it followed spinal as well as inhalation anesthesia. Furthermore, they reasoned that the most likely cause was obstruction of the bronchioles by inflammatory edema and secretion, these secretions blocking the tubes easily on account of lessened ventilating current resulting from weakened diaphragmatic action. In spite of all this, general recognition of its importance and frequency was slow to spread. Likewise, the part played by the various etiologic factors involved in its production, as well as its actual mechanism of production, was even slower to be appreciated. The complete solution of the problem was destined to await the observations and researches of many workers, so that all the factors could be properly evaluated and correlated. The outstanding researches of Coryllos revealed the decisive role played by bronchial obstruction in these cases. Coincidentally, clinical observations by Chevalier Jackson[39] led him to point out the similarity of the symptoms and x-ray pictures in these cases and those of bronchial foreign bodies.

INCIDENCE

Coryllos[18] cautions us to remember that statistics are incomplete on account of the frequent failure to diagnose atelectasis. Notwithstanding the fact that it is the most common postoperative pulmonary complication,[1, 56] it is often overlooked or unrecognized, and is often diagnosed as postopera-

tive pneumonia and reported as such;[40] therefore, much confusion exists in the reported incidence of both conditions. Most reports include them combined in one group. However, this need not perplex us, for, as will be described later, pneumonia is a sequel to atelectasis, so that the whole group is atelectasis in the beginning. At hospitals, where an effort is made to differentiate between these two complications, the incidence of postoperative atelectasis rises while that of postoperative pneumonia falls, and, the greater the effort employed, the greater the incidence of atelectasis becomes.[51] In enumerating these cases, a further source of error probably results from the diagnosis of some cases of atelectasis as pulmonary embolism, on account of the sudden attack of severe pain in the chest.

Most of the cases of both postoperative atelectasis and postoperative pneumonia occur after abdominal operations, especially those in the upper abdomen.

INCIDENCE OF POSTOPERATIVE ATELECTASIS AND PNEUMONIA

	Abdominal Operations Per cent	Other Operations Per cent
King (cited by Adams[1])	14.3	1.2
Rink (cited by Adams[1])	14.0	1.0
Mandl (cited by Elwyn[27])	6.29	0.7

At the Lahey Clinic the incidence of postoperative lung complications was found to be greater after operations on the stomach than after operations on the gallbladder. Their figures, reported by Sise, Mason and Bogan,[76] are as follows:

	Per cent
Stomach operations	14.8
Gallbladder operations	5.9
Average of these two	7.7

Mandl (cited by Elwyn[27]) reported an incidence of 13.8 per cent following operations on the stomach.

Perrone[63] reported 90 cases in which atelectasis most commonly followed operations in the upper abdomen.

Cutler and Hunt (cited by Churchill and McNeil[15]) found the incidence after various operations to be approximately as follows:

	Per cent
Epigastric operations	8
Laparotomies	4
All operations	2

ETIOLOGY AND PATHOGENESIS

Since most of these cases occur after abdominal surgery, the underlying cause must be some factor, or combination of factors, commonly present after laparotomy; furthermore, these factors must be present to a greater degree after upper abdominal than after lower abdominal surgery; and they must be absent, or present to only a slight degree, after other operations. As a matter of fact, the solution of the problem depends upon the correct correlation of a great many factors which form a complex chain of events leading to an end result, that is, atelectasis. However, one factor is conspicuous above all others, that is, bronchial obstruction. As early as 1878, Lichtheim (cited by Elliott and Dingley[26]) produced atelectasis in rabbits by obstructing a bronchus with a laminaria plug. He also demonstrated that collapse did not follow obstruction if the blood vessels to the lung were tied, thus proving that collapse of the alveoli was not an active, but, a passive process, depending upon absorption of their contained air into the blood stream. The more recent extensive experimental work of Coryllos[17] and his associates has demonstrated the importance of complete bronchial obstruction as the one factor that determines the development of atelectasis. All other factors act as predisposing and favoring causes; they cannot produce atelectasis by themselves, although Burford[12] believes that extremely shallow breathing, by not ventilating the peripheral alveoli, can lead to collapse of these with resulting patchy atelectasis, without the presence of bronchial occlusion. However this may be, complete bronchial obstruction stands out as the one condition which definitely leads to atelectasis. Once this occlusion is established, the air in the alveoli, tributary to the occluded passage or passages, becomes absorbed by the blood; and, since no new supply of air can enter, collapse of these alveoli must follow. The type of ate-

lectasis that develops depends upon the size of the obstructed passage or passages.[21] Obstruction of the smaller bronchioles will lead to lobular atelectasis, while obstruction of a lobar bronchus will lead to lobar atelectasis, and obstruction of a main bronchus will lead to collapse of a whole lung.

Coryllos' observations have been verified clinically many times. Jackson[39] was the first to compare these cases with massive atelectasis which complicates foreign bodies in the bronchi. He noticed the similarity of symptoms and x-ray findings in both groups. Tucker,[81] a member of Jackson's clinic, was the first person to demonstrate bronchial obstruction in a clinical case of postoperative atelectasis. With the use of the bronchoscope he found a plug of thick, tenacious mucus causing complete obstruction in the main bronchus and its subdivisions leading to the atelectatic lung. After removal of the plug the lung returned to normal. Since then he and other bronchoscopists have made many similar observations. Thus complete bronchial obstruction is generally accepted as the immediate precursor of atelectasis.

Having arrived at this conclusion, our next step is to determine the manner in which this obstruction occurs. Theoretically, there are three possible ways in which the bronchial tree may become obstructed:

1. Reflex bronchospasm.
2. Swelling of the bronchial mucous membrane.
3. A plug formed by the presence of normal or abnormal secretions or by any aspirated material.

Reflex Bronchospasm: Some observers, notably Scott,[73] Cutler[75] and de Takats,[25] believe that stimuli arising in the field of operation give rise to a nerve reflex, probably through the vagus or sympathetic system, which produces bronchoconstriction. They believe that all other factors, including the tenacious mucus, are secondary. Even Coryllos (cited by Adams[1]) agrees that a small minority of cases may be due to this mechanism. In experiments on dogs, de Takats[25] observed that vagal stimulation, pulmonary embolism, manipulation of intraabdominal organs, and blunt injury to the chest wall all produce bronchoconstriction and increased bronchial secretion, a combination of conditions that favor obstruction. He also demonstrated that this reaction could be inhibited either by section of both vagus nerves or by the intravenous administration of atropine. He is of the opinion that clinical cases of pulmonary embolism give rise to reflex atelectasis more often than to infarction.

Swelling of Bronchial Mucous Membrane: Such a swelling may be the result of inflammation or of allergy. Allergic reaction as a cause of bronchial obstruction seems to be a possibility in some cases. Brown[11] found an edematous swelling causing obstruction of the right main bronchus in a patient whom he had bronchoscoped. Bergamini and Shepard[9] reported two fatal cases of bilateral total atelectasis, following anesthesia, in which the histological appearance of the alveoli and bronchioles was that of angioneurotic edema. Clarke[16] reported two cases of atelectasis complicating asthma in patients who had not been operated upon. Tucker[81] reported another case and expressed the opinion that the mechanism of obstruction was the same as in postoperative atelectasis. Clarke,[16] in reviewing the literature, found that more than 10 per cent of the reported cases of postoperative atelectasis had allergic histories and tests. In the light of these observations, it is very likely that a certain percentage of cases of postoperative atelectasis, in allergic patients, have an allergic factor, which, if not the actual inciting cause, must at least have its share in the production of the obstruction. The allergic reaction certainly furnishes the thick, tenacious mucus necessary for the formation of a mucus plug.

Mucus Plug: The concensus of opinion is that the usual cause of bronchial obstruction is a plug of thick, viscid and tenacious mucus. Such a plug may be formed from the normal bronchial secretions, from bronchial secretions which result from irritation or allergic reaction, or from material aspirated from the mouth and throat, for ex-

ample, saliva, vomitus, pus draining from infected teeth and gums, pus draining from infected sinuses into the nasopharynx, or blood from upper respiratory operations. However, the mere presence of these fluid substances in the tracheo-bronchial tree cannot produce obstruction, for, as Coryllos[18] has said, "Bronchial obstruction cannot be produced unless two factors are present. The first is a more or less viscid bronchial secretion and the second, the inability of the lungs to expel it." Normally the tracheo-bronchial tree rids itself of accumulated secretions or aspirated fluid material by three means:[35]

1. The cough reflex.
2. The action of cilia.
3. Peristaltic movements in the air sacs, bronchioles and bronchi.

The tidal air passing through the tracheo-bronchial tree also aids by loosening and moving obstructions as it passes.[83] Any condition which interferes with this defensive mechanism will favor the retention and stagnation of secretions. That is exactly what happens in these cases. There is a markedly weakened respiratory force and a weakened or abolished cough reflex. Ciliary action is not abolished, except by excessively deep anesthesia or disturbed blood supply,[49] but its effectiveness becomes greatly reduced by the relatively large volume of accumulating secretions.

Most observers agree that the process is initiated by the pulmonary hypoventilation that results from the diminished respiratory effort. Muller, Overholt and Pendergrass[55] place primary significance upon this hypoventilation. They believe that the effects it produces are the determining factors that lead to obstruction of the bronchi and bronchioles. It acts in two ways to promote stagnation of secretions:

1. It reduces the size and motility of the bronchi.

2. It reduces the volume and force of the air that can be expelled from the lungs by coughing.[34, 55]

The result is the accumulation of normal secretions, inflammatory exudate or aspirated material. Yandell Henderson[37] pointed out that during shallow breathing many alveoli remain unventilated. The accumulating secretions may then obstruct the bronchioles which lead to these alveoli. Churchill[14] claims that even the normal bronchial moisture may cause obstruction if the breathing is shallow enough. Secretions of low viscosity may thus obstruct the finer bronchioles and produce scattered areas of lobular atelectasis,[12] but they could not obstruct the larger bronchi to produce massive atelectasis. Nevertheless, this mechanism may act indirectly to produce massive atelectasis by the spreading and confluence of many neighboring areas of lobular atelectasis. In order to occlude the larger bronchi, Band and Hall[6] by experiments on dogs, have shown that the secretions must have a viscosity corresponding to that of gum acacia of not less than 75 per cent concentration. Through the bronchoscope they saw the viscid material adhere to the bronchial wall. As the air passes through on inspiration and expiration there is a slight to-and-fro movement of the mass. During the dilatation of the lumen that accompanies inspiration the mass gradually moves down until it becomes arrested in a location where the lumen is of smaller calibre. Then partial, and finally, complete obstruction occurs. In clinical cases, according to Coryllos, the bronchial stasis and possibly also the anesthetic vapor or gas cause irritation of the bronchial mucosa. This leads to increased mucus secretion. Then secondary infection of these secretions by micro-organisms, including pneumococci, which descend from the upper respiratory tract, produces a bronchitis, accompanied by inflammatory exudate which becomes more and more viscid because of the production of fibrin by the pneumococcal infection. The resulting tenacious fibrinous exudate, plus inflammatory edema of the bronchi, produces obstruction of a lobar bronchus thus producing lobar atelectasis.

SECONDARY PREDISPOSING FACTORS

In the development of postoperative atelectasis, it is generally acknowledged

that there are two chief factors, as I have pointed out:

1. Pulmonary hypoventilation, which acts as a predisposing cause.
2. Complete bronchial obstruction, which is the immediate inciting cause.

In addition, there are a great many secondary predisposing factors, some of them being so important that our etiologic picture would be far from complete without a clear understanding of the exact role played by each of them. They may be classified into two groups:

I. Those which act together to cause or augment the pulmonary hypoventilation.

II. Those which assist in the formation of bronchial plugs.

I. Predisposing Factors Associated with Hypoventilation.

1. Location and type of operation.
2. Elevation of diaphragm.
3. Reduced excursion of diaphragm.
4. Lowered vital capacity.
5. Increased intrapleural pressure.
6. Air beneath diaphragm.
7. Severity of operation.
8. Amount of postoperative pain, muscle spasm and muscle splinting.
9. Degree of obesity.
10. Intra-abdominal complications.
11. Abdominal binders.
12. Use of sedatives and hypnotics before and after operation.
13. Posture.
14. Old age.
15. Debilitating conditions.
16. Poor operative risks.
17. Sex.
18. Straining respiration.
19. Anesthesia.
20. Surgical technic.

II. Predisposing Factors Associated with the Formation of Bronchial Plugs.

1. Pre-existent pulmonary pathologic conditions, including allergy.
2. Hypersecretion of saliva and tracheobronchial mucus.
3. Preoperative use of atropine.
4. Aspiration of material from mouth, pharynx and nasopharynx.
5. Ineffective cough.

PREDISPOSING FACTORS ASSOCIATED WITH HYPOVENTILATION

One of the most important predisposing factors is the location and type of operation. Most of the cases of postoperative atelectasis occur after abdominal surgery, especially after operations in the upper abdomen.[27, 63] As a matter of fact, it rarely occurs as a complication of other operations.[27] Its incidence even varies in different upper abdominal operations. Sise[76] found it two and a half times as frequent after stomach as after gallbladder operations. During and after laparotomy the diaphragm is always greatly elevated and its excursion is reduced, and the expansion of the chest is diminished. Obviously, the result is a reduction in vital capacity. Powers[65] demonstrated, by actual measurements, that abdominal operations are followed by a marked reduction in vital capacity and that the greatest reduction and most prolonged period of recovery is after operations on the upper abdomen. Muller, Overholt and Pendergrass[55] also noticed this prolonged period of recovery after upper abdominal operations. Sise[76] observed that the operations which cause the greatest reduction in vital capacity are the ones which are followed by the greatest incidence of postoperative pneumonia. Thus it is clear that this reduced vital capacity, which means pulmonary hypoventilation, is an important reason why laparotomy makes the patient so susceptible to atelectasis.

VITAL CAPACITY AFTER OPERATION IN VARIOUS SITES

(The percentages refer to the per cent of preoperative level)

	Operation	Vital Capacity
Powers[65]	Extremities and rectum	No Reduction
Churchill and McNeil[15]	Abdominal	Reduced to 25-50%
Sise[76]	Lower abdominal	Reduced to 30-80%
"	Upper abdominal	Reduced to 12-50%
Head[6, 76]	Lower abdominal	Reduced to 60-70%
	Upper abdominal	Reduced to 12-22%
	Lower thorax wall	Reduced to 66-86%
	Lower extremity	Reduced to 90%
Muller, Overholt and Pendergrass[55]	Upper abdominal	Reduced to 33%

An additional factor involved in the lowered vital capacity is an increase in intrapleural pressure. As a result of experiments on dogs, Prinzmetal, Brill and Leake[66] claim that ether anesthesia makes

the normally negative intrapleural pressure less negative, therefore relatively increased, thereby producing less lung distention or relative lung compression. The intrapleural pressure is further increased by abdominal incision, pressure on the abdomen, and traction on abdominal viscera. They attribute this increase of intrapleural pressure to the elevation of the diaphragm.

Sise,[76] in comparing the x-rays of ten gallbladder cases before and after operation, found the right half of the diaphragm elevated and the adjacent lung tissue compressed. By fluoroscopy he noticed that the excursion of the diaphragm was reduced on both sides but especially on the right. Similarly Muller, Overholt and Pendergrass[55] observed this elevation of the diaphragm in the x-rays of 25 cases of upper abdominal surgery, the maximum range of motion being reduced to one-third of its preoperative range. The tidal excursion was reduced to one-half of its preoperative range and took place in the upper third of the possible range of motion, while before operation it had occurred in the lower third. They also found that roentgenograms made on the first postoperative day resembled those made preoperatively during full expiration. Band and Hall[6] believe that some diminution of diaphragmatic activity may be of reflex origin due to its relation to peritoneum, which exercises an influence over any movement that may be deleterious to itself or its contents. The first one to refer to weakened action of the diaphragm, in connection with atelectasis, was William Pasteur,[59] who, in 1890, reported the occurrence of atelectasis in cases of diphtheritic paralysis of the diaphragm. He believed this paralysis led to the development of atelectasis. However, Jackson,[10] in view of his own experience with diphtheria, believes that these cases were really due to bronchial obstruction by the diphtheritic exudate and not to paralysis of the diaphragm. By removal of these obstructions he prevents atelectasis in his own cases. This, however, is a different kind of case from the one under discussion. In diphtheria, with the rapid formation of firmly adherent and highly fibrinous exudate, no secondary factors are necessary to produce obstruction. It is comparable to the case in which an extrinsic foreign body has become lodged in a bronchus. On the other hand, in the cases under consideration, it is very likely that the weakened action of the diaphragm together with its high position, reducing the volume of the chest, and, thereby, the vital capacity and the force of respiration, sets the stage for the obstruction to develop.

AIR BENEATH THE DIAPHRAM

Overholt[55] reported that in 25 patients who had upper abdominal operations x-ray films of many of them showed air visible beneath the diaphragm. In experiments on dogs,[58] he verified the observations of others (Keppick, Melchoir and Melchio, and Wagoner and Krause) that the intraperitoneal pressure in the upper part of the abdomen is normally subatmospheric. He found that the intraperitoneal pressure decreases during inspiration and increases during expiration. Thus the subdiaphragmatic and intrapleural pressures fluctuate in the same direction during respiration. He suggests, from this observation, "that, normally, free diaphragmatic activity is favored by a subatmospheric pressure in the upper part of the abdomen. It would obviously be more difficult for the diaphragm to contract against a positive intraperitoneal pressure."

Using x-ray films, he observed the effect of: (1) injecting air into the peritoneal cavity and (2) laparotomy. In either case he observed, in every animal studied (1) an elevation of the diaphragm and (2) a diminution in diaphragmatic excursions. The reduction in the amplitude of the excursion amounted to approximately 50 per cent. "Under these conditions the diaphragm must move between a positive intraperitoneal and a subatmospheric or negative intrapleural pressure." Overholt does not claim that all of the diaphragmatic restriction after laparotomy is due to the presence of air in the peritoneal cavity, but he says that it may be partly responsible. This fact is fairly obvious.

SEVERITY OF THE OPERATION

Postoperatively, pain plays a very important part in restricting respiratory movements.[15, 39] The more severe the operation, the greater is the amount of postoperative pain, and the more pronounced are the results of the pain, which are:

1. Voluntary splinting of abdominal muscles in order to minimize pain.
2. Reflex muscle spasm which is even more effective in maintaining immobilization.
3. Voluntary guarding against coughing in order to minimize pain.

The splinting of the abdominal muscles and the muscle spasm markedly restrict the respiratory movements, especially those of deep and forcible expiration, such as cough. This further reduces vital capacity, and the lack of efficient coughing allows secretions to accumulate instead of being expelled. Sise[76] claims that these effects are more marked in the upper abdomen because the tension there is greater than in the lower abdomen, and the tension produced by inspiration is also greater because the costal margins are moving in opposite directions. The resulting greater tendency toward pain produces greater splinting and spasm of the muscles. Capelle (cited by Gius[31]) agrees with this muscle splinting theory and offers as proof the fact that he was able to reduce the spasm by the continuous injection of a local anesthetic into the wound and thus restore practically normal abdominal and thoracic respiration as well as vital capacity.

DEGREE OF OBESITY

Haight and Ransom[34] mentioned obesity as a factor in the causation of hypoventilation. This may act by making breathing more difficult due to the increased intra-abdominal content. If this is a correct view, then the greater the obesity, the greater would be the effect.

INTRA-ABDOMINAL COMPLICATIONS

Intra-abdominal complications, such as distention of the intestines,[34] acute dilatation of the stomach, and acute inflammatory complications, such as general peritonitis, lower the vital capacity. The first two act by encroaching upon the diaphragm. This was demonstrated in a case reported by Brunn and Brill.[12] In their patient, following an appendectomy, distention developed; the x-ray showed a high diaphragm with rotation of the heart; physical examination revealed diminished breathing, dullness and rales at both bases. Following an enema the x-ray was normal and the physical signs had disappeared. Peritonitis acts by increasing pain and distention.

ABDOMINAL BINDERS

Churchill and McNeil[15] showed that "the vital capacity of a normal individual was reduced 30 per cent by the mere application of a tight dressing over the right upper quadrant and costal border with the application of a binder such as is commonly employed to hold it in place." The more inelastic the dressing, the more it hinders respiration. Sise[76] points out that the effect of binders "is more marked in the upper abdomen than in the lower because more of the abdomen is usually covered by them and because in the upper abdomen they cover also the lower ribs."

USE OF SEDATIVES AND HYPNOTICS BEFORE AND AFTER OPERATION

Sedatives, administered before and after anesthesia, act by further reducing the pulmonary ventilation and by depressing the cough reflex. Both the barbiturates and the opiates depress respiration. In addition, the opiates depress ciliary action and the cough reflex, while at the same time increasing vagal tone with its resulting narrowing of the bronchi and increased viscosity of mucus.[10, 32] According to Brace,[10] these effects are especially dangerous in patients who have smokers' bronchitis or some other pulmonary inflammatory disease which depends on the cough reflex for the maintenance of an open airway. He says, "A state of inactivity of the cough reflex induced by drugs given 12 hours before the operation becomes a calamity when the re-establishment of the cough reflex is prevented before the evacuation of the accumulated secretion by additional drug therapy, administered one and one half hours before the operation."

POSTURE

The position of the patient has a definite effect on the ability to breathe. It has been shown that both the recumbent and the Trendelenburg positions embarrass the action of the diaphragm and reduce vital capacity.[6, 12, 75] During operations in which the patient lies on one side, the lower side is splinted by the weight of the body, thus restricting respiratory movements on that side. Secretions gravitate into the lower lung, thus providing the material for the formation of a plug.[12]

OLD AGE; DEBILITATING CONDITIONS; POOR OPERATIVE RISKS

Aged patients and patients with debilitating diseases are already suffering from pulmonary subventilation. They form a group which is highly susceptible to the action of additional factors that further decrease the respiratory effort. Waters[80] and his collaborators at the Wisconsin General Hospital observed that the percentage of postoperative pulmonary complications increased in proportion to the preoperative risk.

SEX

The sexual difference in the manner of breathing seems to predispose the male sex to atelectasis. It is common knowledge that normal respiration in the male is more abdominal than thoracic in type; consequently, after abdominal operations, the vital capacity is reduced more in men than in women.[34] As a result we should expect the incidence of atelectasis to be greater in men. Accordingly, King reported an incidence of pulmonary complications twice as great in men as in women.

STRAINING RESPIRATION

Straining respiration, from too light anesthesia or associated with coughing, grunting, moaning, etc., in which the vocal cords approximate and resist the passage of the expired air through the trachea, aids in the development of atelectasis. By experiments on dogs, Van Allen, Adams and Lindskog[82, 83] showed that straining respiration, in the presence of bronchial obstruction, helped to produce atelectasis, and the rate of atelectasis formation increased with increased straining. The increased pressure, on the air entrapped in alveoli whose airways are obstructed, leads to more rapid absorption.

ANESTHESIA

The anesthetic was for a long time, in fact, until approximately 20 to 25 years ago, regarded as the sole cause of all postoperative pulmonary complications, except pulmonary embolism. The irritating effect of ether, with its consequent stimulation of the secretion of large amounts of bronchial mucus, was well known; therefore, until more was known about other less obvious factors, it was quite natural to suspect the anesthetic agent very definitely. However, that the volatile anesthetic agents cannot be the sole cause is shown by two facts: (1) the occurrence of postoperative atelectasis almost exclusively as a complication of abdominal surgery, and (2) the fact that this complication occurs, after this type of surgery, just as often or oftener with spinal, local or regional block, in none of which direct irritation of the lungs occurs. In this connection, Yandell Henderson[36] calls attention to the fact that the pulmonary irritation caused by war gases leads to edema of the lungs instead of massive collapse. Taylor, Bennett and Waters[80] found the incidence of postoperative pulmonary complications at the Wisconsin General Hospital to be greater with spinal and regional block than with general inhalation anesthesia. Their results are as follows:

	Per cent
Cyclopropane	0.95
Ether, nitrous oxide and ethylene	1.36
Spinal and regional block	1.90

Mandl (cited by Elwyn[27]) reported the following incidence of postoperative pneumonia:

	Per cent
Ether	4.44
Local	4.39

Elwyn[28] reported the following incidence of postoperative pneumonia:

	Per cent
General	3.0
Local	2.7

Erace[10] reported an incidence of atelectasis with spinal equal to that with inhalation anesthesia. Brown (cited by Haight and

Ransom[34]) believes that atelectasis occurs more frequently after spinal than after inhalation or regional anesthesia because spinal inhibits the depth and force of the respiratory movements both during and for a time after the operation. Yandell Henderson believes that there is a diminution of body tonus with all types of anesthesia. With high spinal there may occur motor paralysis up to the fourth thoracic segment, thus producing paralysis of the lower intercostal muscles and resulting in hypoventilation.[72] This condition, acting concurrently with other predisposing factors, which are usually present with this type of anesthesia as well as with inhalation anesthesia, leads to the development of atelectasis.

The figures of Taylor, Bennett and Waters are most favorable to cyclopropane. Likewise, Burford[13] reported a low incidence of postoperative pulmonary complications after cyclopropane. His figures are as follows:

	Per cent
626 Abdominal operations	2.07
707 Non-abdominal operations	None

This percentage is greater than that of Waters and his collaborators but it is smaller than the average percentage at large, which is 4 per cent. Nevertheless, in spite of these favorable results, Burford found that, with cyclopropane, the incidence of such complications has increased more than should be expected when the anesthetic was of long duration (average two hours, one minute), especially for operations on the stomach. In all stomach cases the percentage rose to 21.7. A possible explanation of this occurrence may be found in the opinion of Brace[10] who strongly considers cyclopropane to be the most likely anesthetic agent to predispose to atelectasis "because of its marked parasympathomimetic action and depression of the respiratory center with decreased ventilation, inhibition of the cellular dehydrogenases, bronchial constriction, and increase in thick mucus." He considers ether to be less likely to lead to atelectasis, even though it produces an excess of secretions, because it does not produce bronchial spasm nor respiratory depression, the latter not occurring with ether until the stage of overdose is approached. This bronchial spasm in cyclopropane anesthesia is especially likely to develop when rushing a patient to a deeper level by suddenly increasing the concentration so that it becomes irritating.

When we consider what I have said, concerning inhalation anesthesia, we are almost inclined to let the pendulum swing to the extreme opposite direction, and, instead of condemning it unqualifiedly as was formerly done, to absolve it of nearly all blame. If we consider only the irritating effect of ether or cyclopropane on the bronchial mucosa, this policy might be somewhat plausible; but a careful analysis of other anesthetic factors involved forces us to revise our judgment so as to place, not only inhalation anesthesia, but every form of anesthesia in its proper category as an important, although, admittedly, not the only predisposing cause of postoperative atelectasis. These factors may be enumerated as:

1. The rapidity of absorption of anesthetic gases and vapors, as well as the physiological gases.
2. The general loss of muscle tonus.
3. The depth of anesthesia.
4. The duration of anesthesia.
5. The technic of the anesthetist.

THE RAPIDITY OF ABSORPTION OF GASES AND VAPORS

Coryllos[23] demonstrated in dogs that air in an obstructed area of lung takes six to 15 hours to be absorbed, while pure oxygen, carbon dioxide, or anesthetic gas or vapor are absorbed in a few minutes. Nitrogen and other inert gases take a long time to be absorbed (16 hours in the case of nitrogen) thus it is seen that the slow absorption of air is due to its high content of nitrogen. It is also clear that if an obstructed portion of lung contained a mixture composed of a high percentage of anesthetic vapor and oxygen, atelectasis could develop in a very short time following bronchial obstruction. This is what happened in the two cases reported by Bergamini and Shepard,[9] in which massive atelectasis developed soon after the operation. More recently Jones and Bur-

ford[12] reported four deaths from massive atelectasis of one or both lungs, proved by autopsy. These cases occurred rapidly after cyclopropane anesthesia. Jones and Burford explained that "with long continued shallow respirations, certain parts of the lungs, especially at the periphery, may not be ventilated at all. If, during the period of lack of ventilation, the gases in the resting alveoli consist of only absorbable oyxgen and cyclopropane, it is possible that they may be completely absorbed before further ventilation takes place. Patches of atelectasis may thus develop without an obstruction in any portion of the bronchial tree. This process, which may be termed creeping atelectasis, could go on, masked by the high oxygen content of the anesthesia atmosphere, until an entire lobe was involved. The first indication of it would appear when the mask was removed at the end of the operation. The patient would show all of the symptoms of respiratory embarrassment, such as marked cyanosis, restlessness and, in some instances, cessation of breathing." However, if the blood nitrogen of the supplemental and residual air are preserved, or if helium or hydrogen are added to the mixture, the absorption rate is lowered so that atelectasis takes longer to develop, thus allowing time for possible reaeration of the lung.

GENERAL LOSS OF MUSCLE TONUS; DEPTH OF ANESTHESIA

Yandell Henderson[36] has shown that there is a general loss of tone of all the muscles of the body during anesthesia. The resulting relaxation of the diaphragm may be so marked that x-ray pictures may show it to be 7 or 8 cm. higher than its normal position. Henderson claims, "This is as far or farther than the diaphragm moves under the deepest and most forcibly voluntary expiration" and this "results in such a diminution of the size of the thoracic cavity and such a deflation of the lungs that many minute deaerated areas are produced." He says that this condition is found after more than 80 per cent of all laparotomies when it is sought and that it is pure chance that massive atelectasis does not occur after all major operations, this chance depending upon mucus accumulating and blocking a major airway. He draws the conclusion that postoperative atelectasis, after any form of anesthesia, is due to diminished body tonus and its consequent deflation and collapse of the lungs. The deeper the anesthetic, the greater is the effect on respiration. This is probably the reason why operations which require deep anesthesia are followed by the greatest incidence of atelectasis. A. E. Guedel (cited by Burford[13]) expressed the opinion that 15 minutes of lower third plane anesthesia is more depressing than two hours of first plane anesthesia. In deep third plane anesthesia the intercostal muscles are paralyzed and are, therefore, sucked in during inspiration.[66] Of the thirteen cases of postoperative pulmonary complications reported by Burford[13] at least eleven of them had been brought to deep third stage anesthesia during the course of the operation. Similarly, high spinal has a greater incidence than low spinal,[36, 80] because, in high spinal, the lower intercostal muscles are paralyzed.

Burford is of the opinion that cyclopropane has an important advantage in that it can only rarely produce muscular relaxation beyond a certain degree; thus the degree of diminished muscle tonus is automatically limited.

Taylor, Bennett and Waters[80] reported the following results:

Anesthetic Plane	Percentage of Postoperative Respiratory Complications
General	
Analgesia	5.9
I	7.7
II	7.7
III	11.0
IV	15.0
Spinal	
Low (with intercostal activity)	6.1
High (with intercostal paralysis)	15.5

DURATION OF ANESTHESIA

The duration of anesthesia is a more important factor than is usually realized. The longer anesthesia lasts, the longer is the duration of the reduced muscle tonus with its deleterious effects, and the more opportunity there is for secretions to collect.[72]

and for gases and vapors to be absorbed from alveoli whose bronchioles or bronchi are obstructed.

Taylor, Bennett and Waters[80] reported the following observations:

Duration of Anesthesia	Percentage of Postoperative Pulmonary Complications
Less than 30 minutes	2.9
From 31 to 60 "	7.7
From 61 to 90 "	13.2
From 91 to 120 "	15.2
From 121 to 180 "	19.3
From 181 to 240 "	30.3
Over 240 "	33.3
No data on time	4.6

TECHNIC OF THE ANESTHETIST

There is no doubt that the technic of the anesthetist is of especial significance since many factors, under his control or supervision, are involved. A badly administered anesthetic, with straining respiration, obstructed airways, coughing, and retching, will favor the development of atelectasis. Reduction of the carbon dioxide content of the anesthetic mixture to too low a percentage, by means of the carbon dioxide absorber, will produce shallow breathing. Bronchial irritation, from excessive concentration of ether vapor or cyclopropane, will play a minor role, but, nevertheless, its effect will be added to that of the other factors. This irritation may produce a bronchitis,[27] which is a link in the chain of events that leads to atelectasis. This irritation can be kept at a minimum by the well trained anesthetist. Keeping a patient too long at excessively deep levels, instead of permitting him to return to lighter levels compatible with the surgical requirements, will prolong unnecessarily a high degree of diminished muscle tonus. However, in spite of all that has been done, these complications continue to occur even where skilled anesthetists are employed.[71]

SURGICAL TECHNIC

No less important than the anesthetic technic is the surgical technic. Carelessness on the part of the surgeon or his assistants can do much harm. Large, traumatized wounds, infected wounds, excessive traction on abdominal viscera, rough retraction on abdominal muscles, especially upward retraction that makes pressure on the liver and diaphragm, intra-abdominal packs which interfere to some extent with the descent of the diaphragm, leaning and resting on the patient's abdomen and chest,[66] and dressings that restrict respiratory movements all take part in the production or maintenance of hypoventilation.

PREDISPOSING FACTORS ASSOCIATED WITH THE FORMATION OF BRONCHIAL PLUGS

Pre-Existent Pulmonary Pathologic Conditions: The preoperative presence of inflammatory diseases is not necessary for the development of atelectasis. Scott[73] reported their absence in most of his cases that had been checked by physical examination and x-ray before operation. Nevertheless, the presence of a pulmonary infection will favor the production of excessive thick secretions.

The manner in which allergy favors atelectasis has already been described in the discussion of obstruction of the bronchial tree by swelling of the mucous membrane.

Hypersecretion of Saliva and Tracheo-Bronchial Mucus: Ether and cyclopropane cause a hypersecretion of saliva and tracheo-bronchial mucus. The former may find its way into the tracheo-bronchial tree by aspiration.

Preoperative Use of Atropine: Atropine has been accused of concentrating the secretions so that they become thick, viscid and tenacious[50, 56] and are then difficult to expel. On the other hand, it has been credited with beneficial effects. Brace[10] claims that it abolishes many of the adverse effects of morphine, especially the increased bronchial muscular tone, the secretory action and the depression of the respiratory center. Likewise, de Takats[25] claims that it inhibits autonomic reflexes and thereby prevents the occurrence of bronchospasm.

Aspiration of Material from Mouth, Pharynx and Nasopharynx: Aspiration is probably an important source of obstructing material. Schlueter and Weidlein[71] believe that this occurs very often under general anesthesia. Lemon,[49] in experiments on dogs, observed that aspiration of material in the mouth occurred, whether anes-

thesia was light or deep, when the animals were inclined with head up and even in the horizontal position. As the inclination proceeded, with the head end moving downward, less and less aspiration occurred, until the marked Trendelenburg position was reached, when none occurred. Other experiments showed that aspiration occurs also when large amounts of fluid are suddenly vomited. In the same way, aspiration of infected material, such as pus draining from infected sinuses into the nasopharynx, may occur. Similarly, blood from operations in the nose or pharnyx, may be aspirated.

When a patient lies on one side, there is a tendency for secretions to collect on the dependent side.[12] They may pass from the upper to the lower lung or even from one lobe to another of the same lung.

Ineffective Cough: Cough may act in one of two ways: (1) it may clear the tracheobronchial tree by bringing about the expulsion of secretions or other material, (2) it may drive them deeper so as to favor obstruction. During the act of coughing, the inspiratory rush of air may carry material deeper; and the expiratory phase may force some of it deeper, or spread it to other areas of the lung.[5] It is only material in the larynx, trachea, or larger bronchi which can be expelled by coughing; and then, only if the force of expulsion exceeds that of adhesion. Material below the level of the larger bronchi tends to be forced deeper into the lung.[82, 83]

CORRELATION OF ETIOLOGIC FACTORS

All available evidence demonstrates the fact that postoperative atelectasis is the result of a sort of chain reaction, involving a relatively large number of etiologic factors, the most important of which are pulmonary hypoventilation and bronchial obstruction. Both of these are present in every case, with the possible exception of a few cases in which the mechanism may be that described by Jones and Burford, and in which atelectasis in the peripheral alveoli follows hypoventilation without actual obstruction. In the vast majority of cases, however, there is obstruction by a mucus plug, the removal of which relieves the condition. Thus it is evident that bronchial obstruction is the actual inciting cause and hypoventilation is the factor which sets the stage for the obstruction to develop. In turn, these two factors are produced by the collective action of a number of the other factors, acting in unison or consecutively. All of these secondary factors will not be found in any one case, but enough of them are always present to induce the undesired result.

COURSE OF EVENTS AFTER DEVELOPMENT OF ATELECTASIS

Once atelectasis is established the further course depends upon the elimination or continuation of the obstruction. If the obstructing mucus is expelled by coughing, or by bronchoscopy the lung will become reaerated and the condition will thereby be terminated. If coughing only partially removes the obstruction the lung may become partially aerated. It is also possible for coughing to clear some bronchioles or bronchi and, at the same time, to spread mucus to others, resulting in newly obstructed areas, thereby reaerating one portion of lung and causing atelectasis in another portion. In like manner, a previously reaerated area may again become atelectatic. Thus the physical signs may change according to the change in location of the obstruction.[18]

If the obstruction is maintained the result will be comparable to that which occurs when the duct of a glandular organ is obstructed,[23] and the course will depend upon the presence or absence of micro-organisms in the obstructed bronchus.[17] If there are no microbes, the case will remain as simple atelectasis. If infection is present, the micro-organisms will invade the collapsed alveoli, and the further course will be determined by the kind of micro-organisms present and their virulence. Many patients have a non-virulent type IV pneumococcus in their respiratory tracts. If this is the only organism present, simple atelectasis will still result, as explained by Coryllos. If the pneumococcus is more virulent the exudate will be richer in fibrin, and there-

fore more viscid and more difficult to dislodge. The result will be the development of a pneumococcic cellulitis in the alveoli, and the degree of toxicity of the disease will be determined by the number and virulence of the organisms present.[19] If the virulence is low, a mild pneumonia will be the result; and if it is high, a severe pneumonia will result. The variety of pneumonia that develops depends upon the type of atelectasis present. Lobular or patchy atelectasis leads to bronchopneumonia and lobar atelectasis leads to lobar pneumonia.[18]

If, instead of the pneumococcus, the infection is by other bacteria, such as *B. influenzae,* staphylococcus or streptococcus, which produce less viscid exudate, the resulting condition is more likely to be lobular atelectasis followed by lobular pneumonia.[22] If the infection is by a highly virulent pneumococcus the resulting condition is more likely to be lobar atelectasis followed by lobar pneumonia.

Coryllos[21] is convinced that the mere presence of infection in the tracheo-bronchial tree is not sufficient for the production of pneumonia; but that it may result in bronchitis, the exudate of which must occlude a bronchus in order for pneumonia to develop. Thus pneumonia develops as a complication of atelectasis; and postoperative atelectasis and postoperative pneumonia are two phases of the same condition. Elwyn[27] and Band and Hall[6] also are in accord with this conception of postoperative pneumonia.

Furthermore, the experimental and clinical observations of Allen,[3] Joannides,[41] Smith,[77] Brunn and Brill[12] and Jackson[38] indicate that if the atelectatic alveoli contian anaerobic organisms (spirochetes, fusiform bacilli, streptococci, and vibrios) lung abscess may develop as a complication.

SUMMARY

1. Atelectasis is the most common postoperative lung complication.

2. It is most frequent after abdominal operations, especially those in the upper abdomen; and rare after other operations.

3. It is very often diagnosed as postoperative pneumonia.

4. It is the precursor of postoperative pneumonia and some cases of postoperative pulmonary abscess.

5. It is initiated by a great many interacting factors which lead to the production of pulmonary hypoventilation and bronchial obstruction. The hypoventilation sets the stage for the formation of a mucus plug.

6. Hypoventilation is increased by postoperative pain, muscle spasm, muscle splinting, obesity, tight abdominal dressings and bindings, intra-abdominal complications, the use of sedatives and hypnotics before and after operation, old age, debilitating conditions, the horizontal and Trendelenburg positions, and deep and prolonged anesthesia.

7. The formation of an occluding plug is favored by the preoperative presence of inflammatory or allergic pulmonary disease, hypersecretion of saliva and tracheo-bronchial mucus, thickening of bronchial secretions by the preoperative use of atropine, aspiration of material from the mouth and throat, and ineffective cough.

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MALARIA

AN EPIDEMIOLOGIC STUDY*

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Major, M.C., A.U.S.

Concern has been expressed by both medical and lay groups that the return of troops fron endemic malarious areas would react unfavorably upon the health of continental America. This point of view is mainly based upon the assumption that returning service men may introduce the disease into areas of this country in which malaria has been absent for many years; and too, augment its incidence and severity in other areas where it is normally endemic.

To examine this contention, it seemed feasible to undertake an epidemiologic survey in an area where malaria vectors exist, and where a rigid military control program is in operation. Then, under controlled environmental conditions of camp life, factors which might augment the malarial rate for this community, could be ascertained.

The material for this study was obtained from the medical admissions to the Station Hospital Annex, at the Prisoner of War Camp, Camp Livingston, Louisiana. The period surveyed was from 7 July 1943, the date of arrival of the prisoners of war, to 7 July 1944. The personnel of this camp included German prisoners of war, captured in the North African and Sicilian theatre of operations, and enlisted men of the guard detachment from various parts of continental United States. The prisoners of war had all received malarial fever suppressive therapy (0.1 gm. of atabrine per day) during their tenure of service in their respective areas. No suppressive treatment was given during the period of study.

STATISTICAL DATA

A. Number of patients having proven malaria: 148 (3.9 per cent of the mean prisoner of war strength.)

B. Number of attacks, with period of hospitalization: 272. N. B. 46 patients having a total of 50 attacks of so-called "fever of undetermined origin" (clinical malaria but not demonstrated by blood smear) in the period under study were not included in this analysis.

C. Percentage of malarial fever cases from total admissions to the medical service: 37.7.

Percentage of malarial fever cases from

*The source material for this paper was gathered during the author's tenure of service at the Station Hospital Annex, Camp Livingston, Louisiana.

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total admissions to the hospital (medical and surgical) : 25.5.

D. Origin of infection in those giving a prior history of malarial fever:

North Africa: 30
Sicily: 4
Russia (Crimea) : 2

Total number of attacks outside continental United States: 115.

E. History of two or more attacks: 64

1. Maximum number of attacks in one soldier: 6

2. Average interval between attacks: 56 days

(a) longest interval period: 205 days
(b) shortest interval period: 18 days

F. Distribution of attacks by months.

July, 10; August, 10; Sept., 27; Oct., 17; Nov., 10; Dec., 9; Jan., 18; Feb., 30; March, 45; April, 48; May, 34; June, 14.

G. Most recent primary attack of malarial fever: 5 July 1944.

H. Type of Plasmodium demonstrated: P. vivax. N. B. In three patients P. falciparum was demonstrated: however, in subsequent attacks P. vivax was found to be the causative agent.

I. Average days of fever during period of hospitalization: 2 days.

J. Patients showing liver enlargements: 3; patients showing spleen enlargements: 19.

K. Malarial fever admissions to the Station Hospital, Camp Livingston, Louisiana. "Station Hospital serves only American Personnel in Camp Community."

(1) Total admissions in the period under study: 23,221.

(2) Total number of acute malaria (primary attacks) : 11.

(a) origin of infection, Louisiana area: 8.

(b) origin of infection outside the continental United States: 3.

(3) Total number of chronic malaria (relapses) : 51.

(a) origin of infection: Louisiana area: 6.

(b) origin of infection outside the continental United States: 45.

DISCUSSION

The possible introduction of more virulent strains of malarial parasites from large numbers of service men, coming from hyperendemic areas might offer a serious threat to public health in this country. A perusal of our data for the year studied, shows that despite the presence of vectors for the transmission of the disease (K) there were no sporadic outbreaks of malarial fever, either amongst the prisoners of war, guard detachment, or the surrounding camp community. This is substantiated by our data (A & D) relative to the occurrence of malaria among the prisoners of war (3.9 per cent) who served in moderate to highly endemic malarious areas, and the admissions for malaria fever at the Station Hospital which serves the American personnel (K). Indeed, not one enlisted man affilated with the guard detachment developed malarial fever in the period of study. The author is of the opinion, that the rigid supervision over malarial control practiced at the camp and the surrounding areas, prompt diagnosis and treatment, contributed to keep the malaria rate under control.

Of the 148 prisoners of war studied, 36 gave a history of having had malarial fever outside the limits of the continental United States. (D). When questioned, these prisoners could not state with certainty whether the diagnosis was corroborated by blood smears. However, they manifested clinical improvement under atabrine medication. Though the incubation period of malaria may vary with the type of plasmodium, the resistance of the individual, it is stated to be as early as 10 days, and may extend to seven months (Craig and Faust, Clinical Parasitology, pages 205-206). In our series (G), the most recent primary attack of malarial fever occurred 5 July, 1944. It cannot be stated with certainty that even this attack (prisoner of war) had its origin in this area, for delayed primary attacks may occur as long as a year after cessation of suppressive medication.

Following diagnosis, the patients were put uniformly under routine malaria management as suggested by S.G.O., Circular

Letter 153, 5b, dated 18 August, 1943. None of the patients under study showed untoward effects to atabrine therapy. In general, our experience with atabrine medication (B,C,D,E,F,I,J) confirms the fundamental disadvantages inherent in the available drug therapy (atabrine), namely (1) drugs cannot be relied upon to prevent the development of malaria following the bite of the infected mosquito; (2) they do not eradicate the disease. The taking of drugs serves to check the multiplication of the parasites sufficiently, so that most protected persons will not develop active malaria during the period which they are ingested.

SUMMARY AND CONCLUSIONS

An epidemiologic study was undertaken of 272 attacks of malarial fever in 148 prisoners of war (mean prisoner of war camp strength ±5000) for a period of one year. The analysis revealed that there were no sporadic outbreaks of malarial fever either amongst the prisoners of war, guard detachment, or camp community. In fact, not one enlisted man of the guard detachment contracted malaria in the period under survey. One primary attack of malarial fever in a prisoner of war, occurring 5 July, 1944, may have had its origin in this area, although this too may be a point of contention. The low incidence of malaria in this area is attributable to the malaria control program practiced at this military installation, rapid diagnosis and treatment.

Where rigid control is exercised, the disease is kept local and transient.

Though the utopian achievement of control and eradication of the disease has not taken place, one may be reasonably optimistic that the new knowledge gained in this war in technic of mosquito control and treatment will tend to clear up much of the disease already existing on this continent.

Grateful acknowledgement is made to Major Henry C. Brown, M.A.C., Registrar, Station Hospital, Camp Livingston, Louisiana, and to the Louisiana State Dept. of Health in the preparation of this article.

SPINAL EPIDURAL ABSCESS

REPORT OF A CASE*

J. A. COLCLOUGH, M.D.†

NEW ORLEANS

Acute spinal epidural abscess constitutes a grave surgical emergency. Unrecognized and without early surgical intervention it produces compression, interruption of function, and irreversible damage to the spinal cord. The results of such a transverse myelitis are paraplegia, retention or incontinence, decubitus, infection and death. Those of us who have not seen the development of these consequences should keep the possibility of spinal epidural abscess in mind when examining a patient with a possible meningitis, acute poliomyelitis or encephalitis.

According to Boyce,[1] all available cases of spinal epidural abscess were analyzed by Dandy[2] in 1926. Dandy reported 27 cases, including one of his own. Four of these cases were operated upon and two survived. All other cases were fatal. In 1933 Bunch and Madden[3] studied 19 additional cases and reported two of their own. Three of these were not operated upon and died. Of 16 operated upon, four died, four were left with paraplegia and eight made complete recovery. Echols[4] has reported four cases with recovery in three of the four. To the best of my knowledge, the latest written upon the subject is an article by Grant[5] in June of 1945, in which he reports 14 cases and states that over 200 cases are reported in the literature. I do not believe that this comparatively rapid increase in number represents an increase in incidence, but, rather, that it means increasingly early diagnosis and treatment. In spite of recognition as early as 1820,[6] and since there is only a modest number of such cases available in the literature, a great number of deaths must have resulted from unrecognized acute spinal epidural abscess since

*Read before the Scientific Meeting of the Orleans Parish Medical Society, June 10, 1946.

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that date. The object of this report is therefore twofold: (1) to increase our consciousness in regard to this clinical entity, and (2) to add one more case to the literature.

CASE REPORT

N. G., aged 16, a white male, was admitted to the Charity Hospital of Louisiana in New Orleans on November 11, 1944 with the provisional diagnosis of poliomyelitis. His chief complaint was pain in the back and in the right side.

The family history and past history were irrelevant. The review of systems was negative except for the symptomatology of his present illness.

Present Illness: Fifteen days prior to admission he had developed fever and pain in his back and right side. There were no other symptoms. Neurologic examination was said to have been negative two days previous to the date of admission, but upon admission he had developed muscle spasm and paralysis of the right lower extremity. He had been vomiting and was incontinent.

Physical Examination: The temperature was 100.2° F. The patient did not appear to be acutely ill, but lay flat in bed without moving. He was rational and cooperative. The head was negative. He was unable to flex his neck because of the pain it caused in his back. The neck muscles themselves did not seem spastic. The heart and lungs were negative. Slight rigidity was present in the abdominal muscles, and the bladder was greatly distended. There was slight urinary incontinence. The extremities were negative except for the right lower extremity which was flaccid and externally rotated. There was complete paralysis of the right thigh and leg except for a slight degree of abduction, adduction, internal and external rotation. The abdominal reflexes were absent. The tendon reflexes of the upper extremities were normal. The left patellar reflex was hyperactive, and the right patellar reflex was hyperactive. Both ankle jerks were hyperactive, with slight clonus on the right. No positive Babinskis were found. Kernig's sign was present bilaterally. The blood count was 30,500 white blood cells with 93 per cent polymorphonuclear leukocytes. The impression of the examining physician was acute poliomyelitis.

On November 13, 1944, two days after admission, a spinal tap was done, the level not being recorded. The fluid dripped very slowly and about 1 c.c. of xanthochromic fluid was aspirated which clotted immediately when put in a test tube. The protein content was reported to be 1080 mg. A request for consultation to neurology was answered by Dr. Walker Thompson with the following findings: "Paralysis of the right leg, spasticity of the back, hamstring and other muscles bilaterally, weakness of the right arm, hand, absent abdominal reflexes, incontinence of urine and feces. The cell count done in Lafayette, Louisiana Charity Hospital was 94. I do not believe the patient has poliomyelitis. There is evidence of pressure caused by a lesion apparently located somewhere between the fourth and seventh thoracic segments. Impression, possible epidural and sub-dural abscess. Patient should be seen by neurosurgery. Decompression may be necessary."

The patient had previously been seen by physicians on the orthopedics service, who regarded the diagnosis of poliomyelitis as questionable. Packs over the neck, chest, abdomen, upper and lower extremities were advised. It was also suggested that the patient be watched for signs of bulbar involvement.

Shortly thereafter the patient was seen by me and the following findings were recorded: There was a history of pain in the right side beginning ten days ago accompanied by fever. There was weakness of the legs with rapidly advancing symptoms of cord involvement for the past four days; namely, weakness of the lower extremities, bladder and rectal disturbance (retention and incontinence), weakness of both upper extremities. The biceps reflexes were present and equal; the triceps were absent. The knee jerks were absent and the ankle jerks were present and equal. I was unable to elicit any plantar reflex. There was rather marked stiffness of the neck. Exertion of pressure over the spinous processes from approximately the fifth thoracic to the ninth thoracic produced pain locally which was referred to the chest anteriorly. Deep sensation was partially absent in the lower extremities. There was no clear-cut sensory level, but an area of hypesthesia was noted in dermatomes T 6 to T 10. Attempted spinal puncture at the third lumbar interspace was unsuccessful, and at the second lumbar interspace aspiration resulted in withdrawal of a small amount of blood-tinged, purulent material. A diagnosis of spinal epidural abscess was made and the patient was transferred to the neurosurgical service immediately.

Operation: On November 13, 1944, under general anesthesia, a dorsal midline incision was made from the spinous process of the fourth to that of the eighth thoracic vertebra. The soft tissues were bilaterally separated from the spinous processes and laminae by means of periosteal elevator and sharp dissection. The wound was held open by means of self-retaining retractors. The spinous process and laminae of the sixth thoracic vertebra were removed, and as the ligamentum flavum was being removed, creamy-white pus which was thick in consistency was encountered. There was no odor. The spinous processes and laminae of the fourth and fifth, seventh, eighth and ninth thoracic vertebrae were then removed and the abscess was uncovered throughout this extent. There was no granulation tissue noted. Pus filled the entire area. The limits of the abscess had not yet been reached. By means of a catheter passed epidurally, an attempt was made to determine the extent of the abscess, but judging from the fact that pus was

obtained in this manner for a distance of about three inches beyond the unroofed area both approximally and distally, it was deemed inadvisable to attempt to uncover the entire extent of the abscess. Epidural rubber tissue drains were placed above and below the exposed area, and throughout the extent of the uncovered dura. Following this, the soft tissues were approximated loosely using cotton as interrupted sutures. The operation was two hours and two minutes in duration.

Penicillin therapy was instituted postoperatively and was given through Nov. 23, 1944. Fever persisted until Nov. 17, (four days postoperatively) when the temperature was almost normal throughout the day. Improvement in motor function was slow. Twenty-six days after operation (on December 9, 1944), he began to feel the desire to defecate and urinate, and could accomplish these acts voluntarily. On December 11, 28 days after the operation, he was able to flex and extend his left leg and foot, and to adduct and abduct the left thigh. Six weeks after operation he was able to flex and extend the right leg and thigh, and could evert and invert the right foot. On the following day, (December 29) he could stand, but was unable to walk without the aid of crutches. He was discharged on this date to the physiotherapy clinic. Cultures of the material obtained by aspiration produced staphylococcus aureus pyogenes. Culture from the abscess showed the same organism. The blood culture was negative. The serology was also negative.

On October 22, 1945, the gait was spastic with the greater amount of spasticity being present in the right lower extremity. There was no appreciable muscular atrophy. The patellar reflexes were exaggerated, the right being greater than the left. The ankle jerks were hyperactive, the right being more so than the left. There was some muscular weakness of the right lower extremity, but there was a remarkable degree of recovery of muscular strength on both sides. The Babinski was bilaterally positive. There was no impairment of pain sensation. Proprioception was unimpaired. The deep muscle sense was somewhat impaired on the left. Touch was unimpaired. The operative wound was healed, but there was one small sinus in its lower third where a buried suture had been removed that morning. Vibratory sense was unimpaired. Sustained clonus was present on the right. The abdominal reflexes were absent.

On April 21, 1946, eighteen months after operation, he was again examined. At that time the gait approximated the normal. He stated that he could walk much better, and was now able to dance. Other than some weakness of the right leg, he had no complaint. Neurologically some spasticity was still present. There was some muscular weakness in the right lower extremity. The knee and ankle jerks were hyperactive. Ankle clonus was present on the right. There was no neurological or functional impairment of the upper extremities.

It is now eighteen months since operation, and I believe recovery has about reached the maximum.

DISCUSSION

There is nothing remarkable about this case except the size of the abscess. From passing the catheter superiorly at the time of operation, I know that it extended superiorly to the level of the first thoracic vertebra. Neurologically the cord was affected to the fifth cervical segment or the level of the fourth cervical vertebra. Because pus was obtained by aspiration at the second lumbar interspace I know that it extended inferiorly to the upper border of the third lumbar vertebra. In other words, the lower part of the cervical, the entire thoracic, and the greater portion of the lumbar epidural space was involved. It is also noteworthy that this patient was operated upon more than 48 hours after he became almost completely paraplegic, yet the degree of recovery is almost 100 per cent.

Lumbar puncture in cases of suspected epidural abscess must be done with the greatest of care to avoid the possibility of a needle which has traversed an abscess penetrating to the subarachnoid space. As soon as the interspinous space is reached aspiration should be attempted. If no pus is obtained, the needle should be inserted successively deeper, a millimeter at a time, and aspiration should be attempted as each new depth is attained. This should be repeated until pus or cerebrospinal fluid is obtained by aspiration. If the former is found, the diagnosis is established; if the latter, the Queckenstedt quickly reveals it.

The diagnosis of spinal epidural abscess is usually not too difficult if one thinks of it as a possibility. There is usually a history of a pyogenic infection somewhere in the body. However we were unable to obtain one in this case. Pain in the back at any level, tenderness over the spine, root pain, fever, elevation of the leukocyte count, weakness of any extremity with the development of upper motor neuron signs form a picture which is quite clear. The onset of weakness of the extremities and bladder disturbance makes spinal puncture mandatory. The

laboratory findings of increased protein and cell count are confirmatory evidence. The existence of block makes immediate operation an urgent necessity.

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DISCUSSION

Dr. Dean H. Echols (New Orleans): Surely this is the most "epidural abscess conscious" county medical society in America. About six or seven years ago Dr. J. O. Weilbaecher called our attention to this entity. A year or two later I brought two postoperative cases of epidural abscess to this platform. Since then one of us has published the accumulated cases from Charity Hospital in one of the surgical journals. I am glad Dr. Colclough took up the torch because epidural abscess is too important a disease to go unrecognized. If the diagnosis is made because physicians are thinking about that possibility the patients can be cured. I thought for a time that the advent of chemotherapy would put an end to cases of epidural abscess but they have continued to turn up from time to time just the same. One of the important points that Dr. Colclough brought out is that we can not rely on chemotherapy alone to cure all infections; prompt and adequate surgery is also frequently needed.

Dr. Walker Thompson (New Orleans): This condition is particularly important because it is a medical or surgical emergency that can be taken care of and can be diagnosed without very much difficulty. Of course sometimes it looks like a cord tumor syndrome, an extramedullary cord tumor, starting out with involvement of the roots and pain at the level of the abscess. Rather rapidly it causes pressure upon the cord and paraplegia. The thing that gives it away particularly is the tenderness over the vertebra which I believe is more marked than in cord tumors. There may or may not be severe meningeal signs although the patient is not going to be prostrated like we see in meningitis. I do not believe I have anything else to add except that we should teach this syndrome since it can be taken care of with good results as we see in this case.

Dr. Colclough (in closing): I wish to thank Dr. Echols and Dr. Thompson, both of whom have been my mentors, for their discussion and kind remarks.

The first case of epidural abscess I saw was shown to me by Dr. Echols and chemotherapy cured that particular patient. We have also seen an occasional case that clinically we were sure was brain abscess, cured by the same means. By the immediate institution of drug therapy, either one of the sulfonamides or penicillin, there will possibly be a decrease of incidence of spinal epidural abscesses which come to operation. We must be conscious of this condition. Bear it in mind and if it does not yield quickly to chemotherapy, surgical intervention must be instituted.

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THE METRIC SYSTEM

The American Medical Association has adopted the metric system entirely, not only for all of its publications but also for its New and Nonofficial Remedies. The Committee on Revision of the Pharmacopeia will do likewise, employing only the metric system. The Pharmacopeia Committee rightly points out that metric doses in the past have been based upon the apothecary dose and its equivalent. As an example, the usual dose equivalent for a half grain was considered to be 32 milligrams and for five grains 325 milligrams. It is the point of good sense however, to use round figures in the preparation of metric system dosage so that instead of 32 milligrams drug manufacturers will use 30 milligrams and instead of 325 milligrams will use 300 milligrams for the actual dosage. The same relationship will be followed by the pharmacopeia.

Many of the older doctors and even young physicians seem to have a fear of the metric system. Perhaps if round numbers are used this fear may disappear. Actually the present day physician uses the metric system almost interchangeably with the apothecary's. The physician will order 500 c.c. of a solution to be given intravenously and then he will put the amount of the drug he wishes to be given in this solution in the apothecary system. In prescribing most of the common drugs the apothecary system is employed, yet when it comes to the using of arsenicals the metric system is the one that is almost universally employed. Of neoarsphenamine a gram more or less will be ordered for the patient and the apothecary system will be completely forgotten.

The metric system of course has so many advantages that they need not be recounted. It is the universal dosage system employed throughout the world. From the action of the A. M. A. it behooves the physician to learn this particular system. Elsewhere in the Journal is published a table of metric dosage with approximate apothecary equivalents prepared by the Committee on Revision of the Pharmacopeia. This may be used for ready reference in prescribing in the future.

DIPHTHERIA MORTALITY

For many years it was the custom of the Journal of the American Medical Association to review the diphtheria deaths in the large cities yearly. However, on account of the exigencies of the war and because diphtheria, in incidence, was diminishing markedly, no figures have been published since

1942 until they appeared in the July 27 number of the Journal this year.

First, a word as to the one city of Louisiana, New Orleans, which is reported upon. There has been an increase in the city in diphtheria mortality from 2.5 per hundred thousand population to 3.4. Only Fort Worth, in the cities in the west-south central states, exceeds New Orleans in its diphtheria mortality.

It should be noted that there are many cities in which there have been no deaths from diphtheria whatsoever. There were 28 such cities.

It should be pointed out that in some of the cities which had a fairly large death rate many of the children who died were imported into the city and were not residents thereof. It furthermore should be observed that there has been an increase in the actual number of deaths throughout the cities of the country. There is a tendency for this disease at the present time to be relatively more prevalent among the higher age groups. This would bespeak the influence of immunization of the small baby and a failure to give booster treatments to the older children at the time that they are admitted to school and from time to time thereafter until they obtain maturity. Doctors have been taught well the importance of immunizing the infant but they should not forget that booster protective treatments should be given from time to time, not only with this particular disease, diphtheria, but also with other diseases in which passive immunity is induced.

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THE NEED FOR HOSPITAL BEDS IN LOUISIANA

Comment has been made in a previous editorial on the large amount of money that is spent by the State of Louisiana for the maintenance of general hospitals. It was pointed out that of most states in the Union, Louisiana pays for medical and dental care in a greater amount per capita than any other state except for several of the small New England states, New York and Colorado. A little over 67 per cent of the total expenditures are spent for the general medical care of sick Louisianians which is almost 25 per cent more than any other state pays from its total expenditure for medical care. It was noted also that some 48 per cent of all hospital income is provided from taxes appropriated by our State Legislature as contrasted with the next state, California, of which only 37.8 per cent of hospital income is derived from taxes. From these figures compiled from "Medical Services by Government" published by the Commonwealth Fund a few months ago, it is obvious that Louisiana has been more than generous in taking care of its sick citizens.

The figures that have been given bring up the question as to the need and necessity of further extension of state supported hospitals in Louisiana. The United States Government is prepared to give untold millions to provide hospitals and hospital care for the citizens of the country as a whole but it must be borne in mind that some states have spent and are spending but little; Alabama for example expends only 47 cents per capita for medical care, while Louisiana spends $1.88. Other sections of the country may need additional hospital facilities but merely because the United States Federal Government offers the opportunity of getting a few million dollars which has to be met with an equal contribution by the state should not make the state authorities grab at this money. The state must put up a very substantial amount but the real cost of hospitalization is the continuous running of the institution, its maintenance and its upkeep. The initial outlay is minimal as contrasted with the ultimate outgo of monies for the maintenance of an institution. Certainly it does not seem right, fair nor just that our taxpayers should be assessed additional taxes in order to provide general hospital care which already is more generous than is given by practically any state in the country.

The addition of tuberculosis facilities may be necessary but again we would question the need for any great outgo of money for such facilities. The Pines Sanitarium in

Caddo Parish, for example, is only 35 per cent occupied. The death rate from tuberculosis in Louisiana has gone down by leaps and bounds from a total of 84.5 in 1930 to 45.3 per 100,000 in 1945. The negro rate has diminished almost 50 per cent as has that of the white rate. Tuberculosis is being conquered and great expenditures would seem unnecessary for the care of patients with this disease.

The state is behind most states in the provisions made for psychiatric services but psychiatric cases are diminishing now that syphilis is being so adequately controlled and other therapeutic measures are proving efficacious in prevention and in the care of the psychiatric patient.

It would seem that the burden of proof is on those who advocate a constitutional amendment to increase our state debt by many million dollars. Already Louisiana's expenditure for general hospital care is extremely liberal; tuberculosis is a disease which is decreasing in incidence and in which furthermore since the introduction of pneumothorax treatment of the disease more and more patients are being treated as ambulatory cases. Merely because the Government offers monies is no reason to seize upon this money with the idea that it will be cheap. In the long run the initial Federal grant will be small compared to the total money that will have to be spent in the future by the state to maintain the contemplated institutions.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 DUES

During November notice will be sent to members concerning dues for 1947 which should be paid in December of this year. Amount of dues will be continued at $25.00 and this with dues for membership in the parish or district society should be sent to the secretary of the component organization.

Total active members in the State Society for 1946 is 1576, of which 251 were entitled to deletion of dues because of military service. These figures show an increase of 152 paying members over number for last year. Dues were deleted for 339 members in 1945 whereas this number was decreased by 88 members this year, indicating return of many of our members to civilian practice.

The State Society has undertaken many new activities during the past few months. The Louisiana Physicians Service, Inc., is now in full operation. News of this project has been carried in previous issues of the Journal and will be found in this issue also. The plan for medical service for veterans has been approved by our Executive Committee and submitted to the Veterans' Administration for confirmation. It is hoped that all details may be worked out for this service without further delay as many requests are being received from veterans interested in the plan. The president has appointed a committee, of which Dr. A. V. Friedrichs is Chairman, to make arrangements for a Council on Public Relations which will be a vital part of our future activity.

The importance of prompt payment of dues for continuation of the many activities of our organization can readily be understood and it is hoped that each member will realize the necessity of cooperation and feel

his responsibility in these undertakings by remitting his dues promptly.

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PREPAYMENT SURGICAL PLAN EXPLAINED TO DOCTORS OF LOUISIANA

The Louisiana Physicians Service, Inc., started issuing prepayment surgical and obstetrical indemnity insurance contracts during the past month and within a short time it is expected some doctors will be approached by the insureds and a request made for surgical service as provided for in the policy certificate. Comments are made in this issue of the Journal to explain to the doctors exactly how the plan will work and also to request the doctors' participation in the plan.

Louisiana Physicians Service, Inc., is the result of efforts by the Louisiana State Medical Society through its Committee on Voluntary Prepayment Medical Service to give to the people of this state a voluntary prepaid medical benefit plan.

The Corporation was organized August 18, 1946 under the Laws of the State of Louisiana pertaining to the operation of a Mutual Industrial Insurance Company.

The company is under the supervision and control of the Commissioner of Insurance of the State of Louisiana and received its Certificate of Authority dated September 30, 1946 from Wade O. Martin, Jr., Secretary of State.

PARTICIPATING PHYSICIANS

Doctors of medicine licensed by the Louisiana State Board of Medical Examiners may sign an agreement with Louisiana Physicians Service, Inc.

The participating physician remains free to act in relation to patients in accordance with the custom in ordinary practice.

Louisiana Physicians Service does not contract to provide medical care nor may a participating physician be compelled to perform his services in any way differing from usual custom, except that the participating physician agrees to accept the fees in the Schedule of Benefits as full payment for services rendered under the Participating Physicians Agreement after determining with the subscriber that his yearly income, in the immediate preceding twelve months did not exceed $2,000, if single; or the combined income of the spouse and family group did not exceed $3,000.00.

The physician will make his usual charge to patients whose income is in excess of the amounts stated. Patients whose income is in excess of the brackets stated will pay that portion of the fee, if any, that remains after payment has been made by Louisiana Physicians Service, Inc., to the participating physician.

The subscriber and/or his dependents may select any doctor of medicine properly registered by a state board of medical examiners and payment will be made in accordance with the indemnity provided for in the Schedule of Benefits, provided the subscriber's surgical benefit certificate is in force at the date of service.

It should be noted that Louisiana Physicians Service, Inc., has no agreement with any but participating physicians.

PARTICIPATING PHYSICIANS AGREEMENT

The Participating Physicians Agreement is the contract between the participant and Louisiana Physicians Service. There are no dues, fees or costs of any type associated with becoming a participating physician. The physician may discontinue his agreement with the company on thirty days' notice as provided in the agreement. All the company wants is the assurance that the physicians of the state will cooperate with the prepayment medical care plan as established and sponsored by the Louisiana State Medical Society.

SUBSCRIBERS SURGICAL BENEFIT CERTIFICATE

I Benefits Provided

The term "surgical and other services," as used herein, is defined to mean any of the following services rendered while the certificate is in force, and not during a waiting period, by a doctor of medicine of the subscriber's choice, to the subscriber, or

a dependent, or sponsored dependent covered by this certificate, except those items shown as exclusions under paragraph F.

Surgical Services

A. Surgical services (operative and cutting procedures for treatment of disease and injuries, and treatment of fractures and dislocations).

Obstetrical Services

B. Obstetrical services for abortion, miscarriage, ectopic pregnancy or delivery, but not until after the certificate has been in force for ten (10) consecutive months as a Family Group contract.

X-Ray Services

C. Diagnostic x-ray for surgical services not in excess of the value of Fifteen Dollars ($15.00) between the effective date of the certificate, and the first anniversary thereof, or during any succeeding twelve (12) month period.

Anesthesia

D. Anesthesia services not in excess of the value of Fifteen Dollars ($15.00), between the effective date of the certificate and the first anniversary thereof, or during any succeeding twelve (12) month period.

Laboratory

E. Laboratory services, but only when necessary for surgical diagnosis, and when not available through other insurance, and then not in excess of the value of Five Dollars ($5.00) for each surgical procedure not a part of a series of operations related to the same cause.

Benefits Not Provided

The following services or materials will not be provided under Louisiana Physicians Service Certificate.

Exclusions

F. "Surgical and other services," as used herein, does not include:

1. Hospital, dental or nursing services.
2. Medicines, drugs, appliances, materials, or supplies.
3. Services for industrial injuries or diseases, or services from any government agency, which are, or to the extent such services can be obtained by the subscriber without cost to him by compliance with laws enacted by a federal, state, municipal or other legislative body.
4. Services for self-inflicted injuries while sane or insane.
5. Benefits in excess of One Hundred Fifty Dollars ($150.00) between the effective date and the first anniversary of the certificate, or during any succeeding twelve (12) month period, for a series of related surgical or obstetrical procedures performed at the same time, or for different surgical or obstetrical procedures arising from the same medical cause.
6. Total benefits in excess of Three Hundred Dollars ($300.00) for all surgical and obstetrical services rendered under the certificate between the effective date and the first anniversary thereof, or during any succeeding twelve (12) month period.
7. Plastic operations for cosmetic or beautifying purposes.

Waiting Periods

The certificate will cover surgical or other services rendered in the following types of cases only after the prescribed waiting period has elapsed:

For maternity cases or conditions of pregnancy, ten (10) consecutive months from the effective date of the certificate as a Family Group contract.

For tonsillectomy, adenoidectomy, hemorrhoids, and hernia, three (3) consecutive months from the effective date of the certificate.

For known preexisting conditions (other than those covered by the three (3) month waiting period above), twelve (12) consecutive months from the effective date of the certificate.

PAYMENT OF FEES

Louisiana Physicians Service will make payment directly to the doctor of medicine who rendered service to a subscriber or dependent listed on subscriber's application, provided the doctor of medicine obtains from the subscriber an assignment of the benefits payable under the certificate. A prepared assignment is contained on the

Physicians Service Report and all the physician has to do is have the subscriber sign the assignment of fees payable.

IDENTIFICATION CARD

The applicant or employed subscriber always receives a Surgical Benefit identification card at the time of enrollment. He is impressed with the necessity of presenting this card to the doctor when service is requested. Unfortunately, some subscribers do not do so.

IT IS THEREFORE NECESSARY FOR THE DOCTOR OR THE DOCTOR'S OFFICE ASSISTANT TO ASK EACH PROSPECTIVE SURGICAL PATIENT IF HE OR SHE IS A MEMBER OF LOUISIANA PHYSICIANS SERVICE. We recommend that the doctor secure the following information from the card.

1. Name of subscriber (The person who signed the original application card for the Plan).
2. Address of subscriber.
3. The name of the patient.
4. The number of the certificate.
5. The group number of place of employment.

This permits prompt identification when the case is reported to our office, and, in turn, facilitates handling and payment to the participating physician.

The fact that a person presents an identification card does not necessarily mean he is entitled to surgical service. In order to assure payment of the benefits the doctor or the doctor's office assistant should write the home office or branch office or telephone the branch office listed on the identification card to determine if the patient is eligible for surgical service.

PHYSICIANS SERVICE REPORT

Upon completion of service to a subscriber or dependent listed on application to physician should complete the Physicians Service Report. The report is a simple one page blank that requires only a few minutes to complete. All of the claim blank "red tape" has been eliminated. The form has been designed for brevity and for convenience of the physiciar

The physician will be furnished with a number of these forms at the outset and additional forms may be obtained by writing Louisiana Physicians Service, Room 103, 1430 Tulane Avenue, New Orleans, Louisiana.

By the physician obtaining the assignment of benefits he is assured of receiving the fees payable under the Schedule of Benefits provided the subscriber is entitled to receive the services performed.

Participating Physicians should bear in mind that the fees established herein cover EXAMINATION, OPERATION and AFTER CARE of all SURGICAL conditions. Aftercare is defined as a reasonable postoperative follow-up period for the type of case in question (as established by experience in local surgical practice) and does not include medical treatment of/for non-surgical conditions which may occur coincidentally with surgery. Care and treatment of medical cases are not covered by this Plan at this time. Contingent on growth of the organization's reserves, the policies in the future are expected to cover all phases of medical service, including perhaps house calls.

SCHEDULE OF BENEFITS

The Schedule of Benefits of Louisiana Physicians Service, Inc., includes the more frequent surgical procedures. The Board of Directors recognizes that it would be futile to attempt to list each possible surgical operation.

The fees listed in the schedule are considered fair average fees for the patients UNDER the SPECIFIED INCOME limits and are accepted by the Participating Physician as COMPLETE PAYMENT FOR HIS SURGICAL SERVICES, to subscriber and dependents eligible for full service benefits. THESE FEES ARE NOT INTENDED TO REPRESENT WHAT THE SURGEON'S TOTAL CHARGE WILL BE OR SHOULD BE TO PATIENTS IN THE OVER INCOME GROUP, BUT ARE THE MAXIMUM REIMBURSEMENTS ALLOWED BY THIS PLAN. Therefore, it is extremely important that the patient and

the doctor agree on the amount of any additional fee before services are rendered.

Services listed under "Obstetrics" are applicable only to Family Group Contracts.

The schedule fixes and limits the payment to be made by Louisiana Physicians Service, Inc., on account of surgical services covered by the Certificate, and payment shall in no case exceed the applicable rate fixed in the schedule.

Surgical procedures not specified in the Schedule of Benefits, but otherwise falling within the terms of the subscriber's Certificate, will be paid for at reasonable prevailing charges, which must be approved by Louisiana Physicians Service, Inc.

SCHEDULE OF BENEFITS FOR SURGICAL AND OBSTETRICAL SERVICES

ABDOMEN:	
Appendectomy	$100.00
Cholecystectomy	150.00
Gastrectomy	150.00
Herniotomy (hernia), bilateral	100.00
Herniotomy, single	75.00
Paracentesis, abdominal	10.00
Other cutting through the abdominal wall for treatment or removal of organs in abdominal and pelvic cavities (unless otherwise specivied below)	100.00
ABSCESSES:	
Carbuncle (massive) excision	$ 25.00
Furuncle, incision and drainage	5.00
AMPUTATIONS:	
Arm, forearm, hand	$ 75.00
Coccyx, excision	50.00
Fingers or toes	25.00
Hip or shoulder	100.00
Thigh, leg	100.00
Ankle, foot	75.00
ANESTHESIA:	
Major	$ 10.00
Major, over two hours	15.00
Minor	5.00
ARTERIES AND VEINS:	
Varicose veins, excision or ligation	$ 50.00
BONES AND JOINTS:	
Arthrodesis, large joints	$100.00
Arthrodesis, small joints	75.00
Arthroplasty, hip, knee, elbow	100.00
Arthrotomy	75.00
Bone Graft	100.00
Laminectomy	100.00
Spinal Fusions	100.00
BREAST:	
Mammary gland, excision of tumor for biopsy	$ 25.00
Mammectomy, simple	75.00
Mammectomy, radical	150.00
CHEST:	
Thoracoplasty	150.00
Bronchoscopy	50.00
Empyema, rib resection	75.00
Pneumothorax, initial induction	10.00
Refills	5.00
Maximum	25.00
Paracentesis	10.00
Phrenicotomy or phrenic avulsion	50.00
DEFORMITIES-CONGENITAL: (Indemnity applies only to children under 18 years of age when covered by a Family Group Contract.)	
Club foot	$100.00
Hairlip and cleft palate	100.00
Luxation of hip	100.00
DISLOCATIONS:	
Reduction of temporo-mandibular	$ 10.00
Dislocation of the spine	100.00
Shoulder	40.00
Elbow, closed	35.00
Elbow, open	75.00
Fingers or toes	5.00
Hip	75.00
Knee	50.00
Ankle	40.00
Collarbone	25.00
EAR, NOSE AND THROAT:	
Nasal Septum (sub-mucous resection	$ 75.00
Laryngoscopy: Removal of foreign body	50.00
Removal of growth	150.00
Esophagoscopy with foreign body extraction	75.00
Tonsillectomy and Adenoidectomy	35.00
Tracheotomy	50.00

Mastoid, simple	100.00
Mastoid, bilateral or radical	150.00
Antrostomy, window	50.00
Puncture eardrum	10.00
Puncture of antrum	5.00
Nasal polyp removal	15.00
EYE:	
Foreign body removed from cornea	$ 5.00
Foreign body removed from inside eye or orbit	100.00
Primary suture of lid wounds	10.00
Iridectomy	60.00
Cataract, extraction	100.00
Cataract, needling	50.00
Detached retina operation	100.00
Removal of eyeball	100.00
Muscle operation	75.00
Operation for perforating wounds of the eyeball	100.00
Glaucoma	100.00
Chalazion	15.00
Dacryocystectomy	75.00
Dacryocystorhinostomy	100.00
FRACTURE:	
Skull, operative	$100.00
Nasal bones	25.00
Maxilla	50.00
Mandible, unilateral	50.00
Mandible, bilateral	100.00
Malar	25.00
Clavicle, closed	25.00
Clavicle, open	50.00
Scapula	50.00
Ribs	5.00
Vertebra	100.00
Humerus, closed	50.00
Humerus, open and fixation	100.00
Radius or ulna	25.00
Radius and ulna, closed	75.00
Radius and ulna, open	100.00
Elbow, simple	50.00
Elbow, open reduction	100.00
Fingers	25.00
Pelvis	75.00
Femur	100.00
Knee cap	50.00
Tibia or tibia and fibula, closed	75.00
Tibia or tibia and fibula, open	100.00
Fibula, closed	25.00
Ankle, closed	50.00
Ankle, open	100.00
Toes, closed	25.00
Toes, open	50.00
GENITO-URINARY:	
Cystoscopy — without radiography	$ 15.00
Cystoscopy — with radiography	25.00
Cystoscopy, operative	50.00
Urethrotomy, external	50.00
Hydrocele	50.00
Variocele	50.00
Nephrectomy	150.00
Orchidectomy	50.00
Prostatectomy (one or more stages)	150.00
Vasectomy	25.00
Removal of tumors or stones in ureter or bladder	100.00
Removal of tumors or stones in kidney, open operation	150.00
Circumcision, new born	5.00
Child to 12 years of age	10.00
Adult	15.00
GYNECOLOGY:	
Operation strictly intra-vaginal or intra-uterine	$ 50.00
Hysterectomy	150.00
Fistula	50.00
Salpingectomy	100.00
Ovarian cyst	100.00
Cystocele or rectocele	50.00
Combined	75.00
Suspension of uterus	100.00
OBSTETRICS:	
(Note: Applies only when subscriber and spouse are enrolled on Family Group Contract for ten (10) months or more.)	
Delivery, normal, including prenatal and post-natal care	$ 50.00
Cesarean section	150.00
Ectopic Pregnancy	100.00
Abortion or miscarriage, including curettage	50.00
RECTUM OR ANUS:	
Operation on rectum or anus	$ 50.00
Hemorrhoids, external, thrombosed, incision	5.00
Resection of rectum	150.00
Hemorrhoidectomy	50.00
TENDON OPERATIONS:	
Ruptured biceps, quadriceps, or tendo-Achilles	$100.00

Repair of other severed tendons...	50.00
THYROID:	
Thyroidectomy	$150.00
Ligation of thyroid arteries not followed by thyroidectomy.....	50.00
GENERAL SURGERY:	
Skin cysts	$ 10.00
Tumors, excision (for biopsy).....	25.00
Wounds, superficial, suturing.....	5.00
Wounds, lacerated, extensive.....	25.00

THE RAGING FIRE

Over a long period of time, the medical profession has been confronted with the threat of having forced upon it Federal bureaucratic control of the distribution of medical care. The principle was accepted that this would be a continuing menace until the American people — 130,000,000 people—fully understood the implication of these moves and had become familiar with the aims, methods, policy and vast achievements of American Medicine under our system of the private distribution of medical care.

The American Medical Association, the Medical Societies, and other advocates of free enterprise have importantly aided in this vast and essential educational effort.

The menace of the centralized control of the distribution of medical care is a danger that might be likened to a raging forest fire.

It is through the efforts of the various Medical Societies that more than fifty prepayment medical care plans are in operation in nearly every state of the nation. The doctors throughout the nation have organized their own plans to provide medical care for the people of the nation at a cost far less to the individual than would be required as a weekly payroll deduction for administration by the federal government. This is your plan.

If you are interested in "putting out the fire that is raging now" rather than in building an edifice to house fire-fighting equipment to be used sometime, somewhere, for some fire;

If you are interested in participating in the actual solution of the difficult problems of the distribution of medical care; interested in avoiding political medicine and preserving the private practice of medicine in the United States—

Then, enroll as a participating physician in the Louisiana Physicians Service, Inc., to provide prepayment medical care for the people of this State. Louisiana State Medical Society is financing the company. All that is needed, now, is the physician's cooperation—enroll now.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Nov. 11. Scientific Meeting, Orleans Parish Medical Society, 8 p. m.

Nov. 13. Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.

Nov. 14. Clinico-pathologic Conference, Touro Infirmary, 12 noon.

Nov. 15. Executive Committee, Hotel Dieu, 8 p. m.

Nov. 18. Hotel Dieu Staff, 8 p. m.

Nov. 19. I. C. R. R. Hospital Staff, 12 noon.
Charity Hospital Medical Staff, 8 p. m.

Nov. 20. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Charity Hospital Surgical Staff, 8 p. m.
The Orleans Society of X-Ray Technicians, 7:30 p. m.

Nov. 22. L. S. U. Faculty Club, 8 p. m.

Nov. 26. Baptist Hospital Staff, 8 p. m.

Nov. 27. French Hospital Staff, 8 p. m.

Nov. 28. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
DePaul Sanitarium Staff, 8 p. m.

Nov. 29. New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

Dec. 2. Board of Directors, Orleans Parish Medical Society, 8 p. m.

Dec. 3. Eye, Ear, Nose and Throat Staff, 8 p. m.
Orleans Parish Radiological Society, 7:30 p. m.

Dec. 4. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Mercy Hospital Staff, 8 p. m.

Dec. 5. Executive Committee, Baptist Hospital, 8 p. m.

Dec. 6. Ochsner Clinic Staff, 8:30 p. m.

Dr. Robert A. Katz attended the 25th anniversary meeting of the discovery of Insulin in Toronto, September 16-18. Dr. Katz presented a paper on Diethyl Ether in the Treatment of Ischemic Gangrene.

Dr. John T. Sanders attended the meeting of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons at Hot Springs, Virginia, September 5-7.

WARNINGS FROM THE BETTER BUSINESS BUREAU

It is suggested that you get in touch with the Bureau if you are solicited by the following organizations:

Louisiana Parishes Safety Council, Inc. (J. E. Slaughter).

Western Tax Council, Chicago.

Navy Mothers Club.

The Club Women's Newspaper.

Make it a habit to check all solicitations or gifts by strangers with the Bureau.

CHANGES OF ADDRESS

Dr. Lucien C. Delery, 1708 Pere Marquette Bldg.
Dr. Bernard A. Goldman, 1510 Amelia Street.
Dr. Mercer G. Lynch, Ochsner Clinic.
Dr. Carl H. Rabin, 1510 Amelia Street.
Dr. Albert L. Braunstein, 206 Medical Arts Bldg.
Dr. Hardee Bethea, 1615 American Bank Bldg.
Dr. James S. Webb, 2908 Napoleon Avenue.
Dr. N. Leon Hart, 710 Pere Marquette Bldg.
Dr. John C. Suares, 319 Richards Bldg.
Dr. M. P. Boebinger, 4427 So. Robertson St.

WELCOME HOME

The following physicians were recently separated from military service:

Dr. Louis I. Tyler, Jr., 7215 So. Claiborne Ave.

Dr. Richard W. Vincent, 1615 American Bank Bldg.

Dr. Richard E. Robichaux, Raceland, La.

CALLED TO MILITARY SERVICE

Dr. Thomas G. Baffes was recently commissioned a first lieutenant in the Medical Corps of the Army of the United States. Lt. Baffes is stationed at the Veterans Administration, St. Cloud, Minn.

CHANGES IN MEMBERSHIP ROLLS

During the month of September the following changes took place in our membership rolls:

Two intern members (Drs. Arthur A. Calix and Herbert deN. Tucker) were transferred to active membership.

One active member (Dr. Daniel M. Kingsley) was transferred to inactive membership because of his temporary removal from New Orleans.

One active member (Dr. Ruth Winston Kope) was removed from membership because of her removal to St. Petersburg, Florida.

One intern (Dr. Paul L. Getzoff) reinstated to membership and transferred to that of active.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

RAPIDES PARISH MEDICAL SOCIETY

The Rapides Parish Medical Society held its regular meeting at the Hotel Bentley in Alexandria on Monday, October 7. The following program was presented: Dr. Chester A. Fresh spoke on psychosomatic problems, Dr. D. W. Oungst discussed the Veterans' Administration and Dr. O. J. Richardson spoke on the organization of visiting staff at Charity Hospital.

RESOLUTION OF TANGIPAHOA PARISH MEDICAL SOCIETY

We, the Tangipahoa Parish Medical Society, feel that certain facts should be called to the attention

of the Louisiana State Medical Society;

A local patient, treated for two years for diabetes as proven by reports of a registered technician, had occasion to visit another physician. He was there informed that he did not have diabetes, but that he had a cancer with only a short time to live.

Feeling that he had been handled in an improper and careless manner, he returned to the local doctor who had treated him, accused him as indicated, then murdered the doctor and committed suicide. Notes found on the patient's person and made public by the coroner revealed this act as a clearly premeditated murder.

We, the doctors of the Tangipahoa Parish Medical Society, wish to express our regret that we have doctors in the field of medicine who do not respect medical ethics and take this means to register our disapproval as this is not the only instance that has come to our attention of a lack of ethics by our colleagues.

Collins P. Lipscomb, M.D.
Secretary

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

A meeting of the Southern Section of the organization was held on Friday, September 27 at the Louisiana State University School of Medicine. The following program was presented:

1. Hematologic Effects of Pteroyl Glutamic Acid in Man. Grace A. Goldsmith (introduced by J. H. Musser), Department of Medicine, Tulane University School of Medicine.

2. Experiments Dealing with the Life History of the Hookworm *Bunostonum Phlebotomum* Parasitic in Calves. Roy L. Mayhew, College of Agriculture, Louisiana State University, Baton Rouge.

3. Differences in Culturability, Infectivity and Pathogenicity of Human Strains of *Endamoeba Histolytica*. Ernest Carroll Faust, Department of Tropical Medicine, Tulane University School of Medicine.

4. An Easy and Accurate Method for Determination of Hemoglobin in Blood, Based upon Its Iron Content from One Drop of Finger-tip Blood. J. T. Peters, Department of Medicine, Louisiana State University School of Medicine.

5. Blood Volume Studies in Congenital Heart Diseases. Woodrow Nelson, H. S. Mayerson, John H. Clark, and Champ Lyons. Departments of Physiology and Surgery, Tulane University School of Medicine.

6. Radiosodium Tracer Studies in Congestive Heart Failure. Paul Reaser and George Burch, Department of Medicine, Tulane University School of Medicine.

PUBLIC HEALTH TRAINING CENTER

The first of October there was opened a Public Health Training Center in the City of New Orleans which is jointly supported by the City and State and which is being given financial assistance by the Commonwealth Fund of New York. The Center will be under the direction of Dr. Ben Friedman who conducted the training center that was supported by the State Board of Health at Franklin, Louisiana, prior to his going into the Army.

The present training center will be a very much more elaborate institution. There will be doctors, trained nurses and sanitarians, as well as health, welfare and social workers. The personnel will be trained both for the City and State Department of Health.

Dr. Friedman was on the staff of General Eisenhower during the war and amongst other honors he received two decorations from the Norwegian Government.

NEWS ITEMS

Dr. Ralph Platou, Professor of Pediatrics at Tulane, addressed the Southern Tuberculosis Conference at Jacksonville, Florida, on "The Management of Primary Tuberculosis in Children."

Dr. Maurice Campagna, of the medical department of Tulane, gave an address on the important subject "Interesting the General Practitioner in Tuberculosis."

Dr. Julius Lane Wilson, president of the State Tuberculosis Association, and Dr. John M. Whitney also were in attendance at this meeting.

Dr. Michael DeBakey of the Department of Surgery, Tulane University, is one of eight members of the new committee called the Medical Advisory Committee, appointed by Secretary of War, Robert P. Patterson.

Miss Mary Louise Marshall, Librarian of the Orleans Parish Medical Society of the Tulane University Medical Library, was one of six speakers who addressed the Association of Honorary Consultants to the Army Medical Library.

The National Gastroenterological Association announces it Annual Cash Prize Award Contest for 1947. One hundred dollars and a Certificate of Merit will be given for the best unpublished contribution on gastroenterology or allied subjects. Certificates will also be awarded those physicians whose contributions are deemed worthy.

The second Inter-American Congress of Radiology will be held in Havana November 17-22. Reservations may be made at the Sevilla-Biltmore. Dr. Leon J. Menville is one of six of the general committee for the United States.

The American Academy of Allergy will hold its annual convention at Hotel Pennsylvania, New

York City, November 25-27. All physicians are cordially invited to attend the sessions as guests of the Academy.

Dr. Myron Wegman, Head of the Department of Pediatrics of the Louisiana State University School of Medicine, attended the Fourth Postgraduate Conference in Pediatrics to be held in Galveston, October 28-November 2. Dr. Wegman addressed the Conference on the topics:

1. Scope diarrhea problems
2. Factors influencing growth and development
3. Common immunization procedures.

Dr. Russell L. Holman, Head of the Department of Pathology and Bacteriology of the Louisiana State University School of Medicine, attended the meeting of the Southern Medical Association in Miami, Florida. On November 5, 1946, he addressed the Section on Medical Education and Hospital Training of Southern Medical Association on "The Integration of the Teaching of the Basic Medical Sciences With The Hospital Services."

Dr. Lyon K. Loomis, Instructor of Orthopedics, also attended the meeting of the Southern Medical Association. His address was on: "Treatment of Simple Oblique and Simple Spiral Fracture of the Shaft of the Tibia."

A splendid opportunity exists for a young man to obtain training in one of the outstanding European hospitals, namely the American Hospital in Paris. Applications for such a position are now being accepted. To the man who may be interested in this position he should take the matter up with Dr. John Archinard of New Orleans.

The American Foundation of Tropical Medicine will establish in Liberia an institute for research in tropical medicine. This undertaking has been made possible through a large gift from Mr. Firestone of the tire and rubber company.

SCIENTIFIC EXHIBIT

Centennial Session—American Medical Association

At the Centennial Session of the American Medical Association to be held in Atlantic City, June 9-13, 1947, the Scientific Exhibit will include both the history of medicine during the past Century and the latest developments of medical science.

Application blanks for space are now available. All applicants must fill out the regular form. Applications close on January 13, 1947, after which time the Committee on Scientific Exhibit will make its decision and notify the applicants.

Application blanks for space should be procured as soon as possible. They are available from The Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

THE NATIONAL CANCER FOUNDATION

This organization will put on its first annual appeal for funds during the month of November. The campaign will be carried on throughout the country. Part of the money obtained from the campaign will be utilized in building hospitals for advanced cancer patients.

CONFERENCE ON GASTRIC CANCER

There will be held December 5-6 at the Billings Hospital, University of Chicago, under the chairmanship of Surgeon General Parran of the United States Public Health Service, a conference on gastric cancer. This is the third such conference that has been held since the original one in 1940. The importance of gastric cancer may be illustrated by the fact that cancer of the gastro-intestinal tract causes 45 per cent of all cancer deaths in this country.

A distinguished group of clinicians, surgeons, pathologists, radiologists, and others will participate in the program.

DALLAS SOUTHERN CLINICAL SOCIETY

The Dallas Southern Clinical Society will hold a postgraduate medical conference November 18-21. These courses are in medicine, surgery, obstetrics and pediatrics.

CAMPAIGN TO COMBAT HEART DISEASE

The American Heart Association announces initiation of a nation-wide program on public education of heart disease. The organization points out that heart disease now kills more than any other disease. In fact the number of deaths due to diseases of the heart is greater than the total of the next five leading causes of death.

The Heart Association will have the support and cooperation of many well known national organizations such as the American Academy of Pediatrics, American College of Physicians and the American Public Health Association.

In pursuance with their purpose of public education there will be held a National Heart Week, beginning February 9, 1947.

The Metropolitan Life Insurance Company is an extremely active participant in this educational campaign. The company has prepared three monographs on heart disease which will be distributed to physicians throughout the country. The monographs are extremely well gotten up and are of interest not only to the physician but primarily to the lay individual. These are prepared for distribution to doctors with the hope that they will place them in their offices or make available to such groups as service clubs, parent-teacher associations and other civic organizations.

NEW YORK RHEUMATISM ASSOCIATION

There will be a scientific meeting of the New York Rheumatism Association on Friday evening, November 22, 1946 at 8:15 p. m., at the Cornell University Medical College Auditorium.

TABLE OF METRIC DOSES WITH APPROXIMATE APOTHECARY EQUIVALENTS

Liquid Measure

Metric	Approximate Apothecary Equivalents
1000 cc.	1 quart
750 cc.	1½ pints
500 cc.	1 pint
250 cc.	8 fluidounces
200 cc.	7 fluidounces
100 cc.	3½ fluidounces
50 cc.	1¾ fluidounces
30 cc.	1 fluidounce
15 cc.	4 fluidrachms
10 cc.	2½ fluidrachms
8 cc.	2 fluidrachms
5 cc.	1¼ fluidrachms
4 cc.	1 fluidrachm
3 cc.	45 minims
2 cc.	30 minims
1 cc.	15 minims
0.75 cc.	12 minims
0.6 cc.	10 minims
0.5 cc.	8 minims
0.3 cc.	5 minims
0.25 cc.	4 minims
0.2 cc.	3 minims
0.1 cc.	1½ minims
0.06 cc.	1 minim
0.05 cc.	¾ minim
0.03 cc.	½ minim

Weight

Metric	Approximate Apothecary Equivalents
30 Gm.	1 ounce
15 Gm.	4 drachms
10 Gm.	2½ drachms
7.5 Gm.	2 drachms
6 Gm.	90 grains
5 Gm.	75 grains
4 Gm.	60 grains (1 drachm)
3 Gm.	45 grains
2 Gm.	30 grains (½ drachm)
1.5 Gm.	22 grains
1 Gm.	15 grains
0.75 Gm.	12 grains
0.6 Gm.	10 grains
0.5 Gm.	7½ grains
0.4 Gm.	6 grains
0.3 Gm.	5 grains
0.25 Gm.	4 grains
0.2 Gm.	3 grains

Weight

Metric	Approximate Apothecary Equivalents
0.15 Gm.	2½ grains
0.12 Gm.	2 grains
0.1 Gm.	1½ grains
75 mg.	1¼ grains
60 mg.	1 grain
50 mg.	¾ grain
40 mg.	2/3 grain
30 mg.	½ grain
25 mg.	3/8 grain
20 mg.	1/3 grain
15 mg.	¼ grain
12 mg.	1/5 grain
10 mg.	1/6 grain
8 mg.	1/8 grain
6 mg.	1/10 grain
5 mg.	1/12 grain
4 mg.	1/15 grain
3 mg.	1/20 grain
2 mg.	1/30 grain
1.5 mg.	1/40 grain
1.2 mg.	1/50 grain
1 mg.	1/60 grain
0.8 mg.	1/80 grain
0.6 mg.	1/100 grain
0.5 mg.	1/120 grain
0.4 mg.	1/150 grain
0.3 mg.	1/200 grain
0.25 mg.	1/250 grain
0.2 mg.	1/300 grain
0.15 mg.	1/400 grain
0.12 mg.	1/500 grain
0.1 mg.	1/600 grain

The above *approximate* dose equivalents have been adopted by the latest Pharmacœpia, National Formulary, and New Non-official Remedies, and these dose equivalents have the approval of the Federal Food and Drug Administration.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending September 22 there occurred in the city of New Orleans 150 deaths which were divided 89 white, 61 colored; 20 of the deaths were in children under one year of age divided equally between the two races. For the following week which came to an end September 14 there was a rather sharp drop in the deaths taking place in the city of New Orleans; 89 of the people that died were white individuals, 33 colored. Only nine were children under one year of age and eight of them were white children. The succeeding week which closed September 21 found 108 New Orleanians expiring, of whom 57 were white and 51 were colored. Seventeen of these deaths were infant deaths with the figures for the previous week practically reversed between the two races. For the week ending September 28 the 122 deaths

in the city were divided 80 white, 42 colored. Seventeen of the deaths were in children under one year of age and this week 13 of these children were white and only four negro. The mortality rate in New Orleans still remains well below that of the previous three year average.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending September 7, showed the following reportable diseases in numbers greater than 10: 72 cases of malaria, 28 of pulmonary tuberculosis, 17 of unclassified pneumonia, 16 of poliomyelitis and 12 of whooping cough. The poliomyelitis cases were pretty well scattered throughout the state. No one parish reported more than two cases except East Baton Rouge with four. For the following week ending September 14, there was again a remarkable diminution in the number of reportable cases. Pulmonary tuberculosis led with 25, followed by poliomyelitis with 21 and typhus fever with 12 instances. Again this week the poliomyelitis cases came from all over the state, but Orleans Parish reported seven and East Baton Rouge four. For the week which closed September 21, malaria was at the head of the list with 54 cases, all contracted within the continent of the United States. Next came poliomyelitis with 20 cases, followed by unclassified pneumonia with 16, septic sore throat with 15, pulmonary tuberculosis 14 and typhoid fever 10. Of the large number of poliomyelitis cases being reported, Orleans Parish and Caddo were the only parishes that had more than two cases. The 10 cases of typhoid fever occurred in 10 separate and distinct parishes. For the week which ended September 28, for the first time, in listing the number of cases in the state of various reportable diseases, pulmonary tuberculosis was the only disease which occurred in number greater than 10, there being 38 patients listed with this disease this week. The epidemic of poliomyelitis seemed to be on the wane with only five cases reported.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending August 31, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	68	410	478
Gonorrhea	1382	11875	13257
Granuloma Inguinale	28	191	219
Lymphopathia Venereum	5	78	83
Syphilis	1799	11961	13760

LAKESHORE HOSPITAL

On June 1, 1946 Lakeshore Hospital located at West End Boulevard and Lakeshore Drive opened. This Hospital was created by the New Orleans Medical Foundation for the purpose of providing sorely needed additional hospital beds for the City of New Orleans and its vicinity. The Hospital is a non-profit institution operated by the New Orleans Medical Foundation, the membership of which consists entirely of veteran medical officers of the armed forces who served in World War II.

Even though the Lakeshore Hospital is operated by an organization composed entirely of veteran medical officers this is not to be misconstrued to mean that membership on the staff of the Hospital is limited to returning medical officers. The actual operation of the Hospital is controlled by a board of directors composed of the members of the executive committee of the New Orleans Medical Foundation plus representatives of other medical groups and hospitals in the City of New Orleans, as follows: the president and the president-elect of the Orleans Parish Medical Society, the president of the New Orleans Hospital Council; and one member of the board of directors of each of the following Hospitals: Touro Infirmary, Hotel Dieu, Mercy Hospital, and Southern Baptist Hospital. In addition to these there are two members at large who were selected by the above members of the Board of Directors.

Even though membership of the staff is open to all doctors licensed in the State of Louisiana who are members of the Orleans Parish Medical Society, applications for membership to the staff are carefully reviewed by the medical committee of the Board of Directors prior to granting the use of the facilities of the Hospital. In effect, this creates an open staff for Lakeshore Hospital which is under the rigid control of the medical committee in order to maintain the existing standards of hospital care in the City of New Orleans.

Lakeshore Hospital is a general hospital fully equipped to take care of all types of medical and surgical cases and has excellent laboratory, x-ray, surgical, and obstetrical facilities. There is also a competent resident staff of doctors capable of assisting the members of the staff in the care of their patients. The Hospital is not equipped to handle neuro-psychiatric problems, terminal care cases, alcoholic cases, or drug addiction cases. If these conditions co-exist with other medical or surgical conditions care can be provided for these types of cases only to the extent where the medical or surgical condition has been brought under control.

The nursing personnel and staff of Lakeshore Hospital is of the highest calibre and experience has demonstrated that in the performance of their duties they are rendering the highest type of nursing service. Miss Imogene Yarbrough is Director of Nurses. The doctors who have had patients at the Hospital have only the highest commendation for the quality of nursing which she has been able to provide. Those on the Board of Directors who have worked with Miss Yarbrough can only express their appreciation and thanks to her interest

and devotion in developing such an excellent nursing staff.

The services though not straight services in the strict sense of the word are divided for the purpose of organization and operation into the following categories: internal medicine, general surgery, orthopedic surgery, obstetrics and gynecology, ophthalmology, and otolaryngology. The staff is similarly organized. The present bed capacity is 150 beds with existing renovations provided for an expansion to 175 beds in the immediate future. Other existing buildings are to be made available should additional beds be needed. The anticipated maximum bed capacity can be expanded to 325 beds. The diagnostic and pathological laboratories are fully equipped and adequately staffed. They are capable of performing all laboratory and diagnostic procedures. The direction of the laboratory is under the supervision of Dr. Friedrichs. It is anticipated that in the very near future a full time medical director of the laboratory will be available at the Hospital for consultation with the doctors and supervision of the laboratory procedures.

In addition to the regular laboratory service is proud to announce that it has the only fully equipped Tropical Diseases Laboratory connected with a private hospital in this section of the country. This Laboratory is under the direction of Dr. Joseph D'Antoni and is available to all members of the staff for diagnostic and consultation services.

The X-Ray Department is under the direction of Dr. J. N. Ané and Dr. H. F. Inderlied. These two doctors will provide 24-hour coverage for the x-ray service. They are available for consultation with the staff doctors at all times. The x-ray equipment is of the best quality and is capable of recording all types of x-ray examinations.

The technical assistants in all laboratories as well as the heads of all departments such as the dietitian and the pharmacist, are registered as certified in their respective fields. Mrs. C. L. Garris arrived at the Hospital on September 13, 1946 and immediately began her work as dietitian for the Institution. In addition, Mrs. Shelby McCaffrey, who is in charge of the Medical Records Library, is a certified Medical Record Librarian. The quality of her work is well known to doctors and her presence assures the Hospital that the records of the patients will be well supervised and duly recorded.

The surgical facilities at Lakeshore Hospital are second to none as existing in the City of New Orleans. The direction of the operating room is under Miss Margaret Bussey who prior to assuming her duties at Lakeshore Hospital was supervisor of the operating room of Southern Baptist Hospital and the 64th General Hospital. The equipment of the operating suite is more than adequate to enable any and all types of surgical procedures to be performed. There are five large, well lighted, well ventilated operating rooms which provide sufficient space to maintain a very heavy daily operating schedule.

The obstetrical wing opened on September 15. The nursery of this service has a capacity at present of 30 bassinets with two incubators. There are four labor rooms and two delivery rooms. The predelivery wing is separate from the post-natal building. Contrary to existing policies of the other hospitals in New Orleans Lakeshore Hospital has not as yet felt that it is necessary to limit post-delivery stay of the patients. It is hoped that if it is so desired all post-partum cases will be able to remain in the Hospital for at least ten days, perhaps in certain cases for a longer period of time.

At this time the Board of Directors of Lakeshore Hospital would like to announce that Dr. W. P. Gardner has been secured as Director of the Hospital. Dr. Gardner assumed his duties as of Monday, October 15. Prior to his arrival Mr. Paul Rutledge, business manager, has been filling the dual role of being both Director and Business Manager. During this period his industry and leadership have made him most successful and it is felt that the arrival of Dr. Gardner will be welcomed by Mr. Rutledge, so that Mr. Rutledge may resume his original position as business manager of the Hospital.

Lakeshore Hospital was opened to help meet the acute bed shortage in New Orleans. The waiting lists for admission to other hospitals in the City continue, indicating that further expansion of this Hospital may be necessary more completely to relieve this emergency. Looking beyond the emergency it is conceivable that the expected increase in population in New Orleans as a result of the shifting population during the War will represent to a great extent a permanent increase in population. In addition to this expanding industry and business will produce a still further increase in permanent population. Present time investigation has shown that additional permanent hospital beds should be added to those already existing in the City. The New Orleans Medical Foundation is cognizant of these conditions and is carefully investigating possibilities for solving this problem, because the members of this Foundation feel that the Foundation can fill a definite need in the medical program not only of the City of New Orleans but also of the Southern States. The ways and means of providing an increase in the permanent beds are being investigated as well as other problems pertaining to the medical care of the citizens of this vicinity. It is hoped that the Foundation can be

not only of moral but also of material benefit in helping to solve some of these problems. With the cooperation of the doctors, and by cooperating with the doctors and other hospitals in the City, this Foundation can be of great assistance in making New Orleans a medical center of the South.

ALFRED B. LONGACRE, M. D.
Chairman Medical Committee.

BOOK REVIEWS

Hypnoanalysis: By Lewis R. Wolberg, New York, Grune & Stratton, 1945. Pp. 342.

There is an urgent need in psychiatry for an abbreviated form of therapy applicable to patients who are unable to avail themselves of prolonged treatment. Existent therapies include suggestion, persuasion, guidance, confession and mental ventilation, reassurance, re-education, desensitization and the correction of remediable elements in the patient's environment that have a destructive effect on his adjustment.

While such therapies are superficial, they are justified because they often eliminate sources of stress and favorably alter behavior that brings the individual into conflict with others. They may also provide a medium for the most effective operation of whatever spontaneous forces of mental health remain and establish the beginnings of more profound psychic change.

Hypnoanalysis has emerged as a procedure contributing speed and directness in therapy. Dr. Wolberg has written the most exhaustive available account of the theory and practice of hypnoanalysis and he has elaborated on the dynamics, indication, limitations, and technic of the method, evolving new experimental procedures. He believes hypnoanalysis has definite values, but will not move mountains and that failures occur as they do with other therapies but he also believes persons respond to hypnoanalysis when other psychotherapies have failed.

With true scientific spirit, the author states "hypnoanalysis holds promise of becoming an invaluable adjunct to psychoanalysis, but it must be explored more thoroughly before its scientific worth can be fully evaluated." The present volume is offered as a contribution in that direction and it is an extraordinarily valuable and important piece of work because human relations, human behavior and human reactions to the desire for security and happiness and the struggle for existence constitute the twentieth century challenge to psychiatry.

C. P. MAY, M. D.

Manson's Tropical Diseases. A Manual of the Diseases of Warm Climates: Ed. By Philip H. Manson-Bahr, C. M. G., D. S. O., M. A., M. D., D. T. M. and H. Cantab, FRCP, London, 12th Ed., Baltimore, Williams and Wilkins, 1945. Pp. 1068, por. illus. Price, $12.00.

The early editions of *Manson's Tropical Diseases* enjoyed almost a monopoly so far as English publications were concerned. Now, however, the situation is different and the twelfth edition comes in competition with several works published since the eleventh edition appeared in 1940. This work differs from some of the newer volumes in that it is the work of a single author whereas several of the other recent publications have been prepared by specialists in the different fields. This edition consists of 840 pages devoted to individual diseases and a few chapters on general subjects, followed by approximately 150 pages of medical protozoology in which the author includes medical helminthology and medical entomology. The latter is followed by approximately 20 pages of laboratory methods. A somewhat unusual feature is the table of drugs for the treatment of tropical diseases. In the preface the author states that many chapters have been rewritten. As a result many of the newer observations have been included. The work has been prepared particularly from the point of view of the clinician with less attention, but perhaps sufficient, to public health and preventive medicine aspects. In comprehensiveness the work stands between the newer concise manuals that have appeared in England and the United States, and the very extensive work of Stitt as revised by Strong. A few points of special interest may be mentioned: The difficulties of absolute differentiation of the cholera vibria from similar organisms are very clearly set forth. The author is impressed by the value of cholera vaccination in the control of the disease, and even is favorably impressed by oral vaccine. Under the treatment of leprosy chaulmoogra oil and its derivatives are given much space but no mention is made of promin, which has attracted so much attention in the United States in recent years. The author takes a very optimistic view of the results of attempts to control leprosy in the Philippines. Not all students of the subject will agree. Opium in full doses is believed by the author to have special virtue in phagedenic processes of tropical ulcers. The author prefers the term Bilharziasis for the parasitic infestation that, in recent years, is more often called Schistosomiasis. We are told that in Egypt mass treatment of cases is being practiced as a prophylactic measure. A few statements may need revision when the next edition is published—thus on page 242 we find the statement that the virus of Rocky Mountain Fever has not

been found in small mamals which is correct, but in Table IV facing page 244, vertebrate reservoirs are mentioned which so far as present knowledge goes is incorrect. On page 248 under geographical distribution of Q Fever the statement is made that it is apparently widespread in America, without making clear that this refers to the distribution of the virus, not of the disease in man. So far as information is available, aside from laboratory infections, but a single outbreak in man has occurred in the United States. In the differentation of the plague organism from that of *Pseudo-tuberculosis rodentium* it is mentioned that rats are instantly killed by *B. pestis*. It is, of course, true that they die in a few days, but instantly is a little misleading. Penicillin is not mentioned under the treatment of yaws. There is no mention of the importance of tracheotomy in dealing with one of the more serious complications of leprosy. In the discussion of general effects of ankylostomiasis it is reported that there may be a predisposition to larceny and other crimes. In general the work is to be regarded as satisfactory and should prove very useful.

G. M. McCoy, M. D.

Narcotics and Drug Addiction: By Erich Hesse, M. D., New York Philosophical Library, Inc., 1946. pp. 219. Price, $3.75.

In this book the author has attempted to cover a wide field extending even beyond that commonly suggested by the title. In addition to the opiates, cocaine, cannabis and so on, which are included under the general heading of narcotics, consideration is also given to alcohol, tobacco and the purine drugs which are classified as stimulants, a rather broad and arbitrary use of the term. On the other hand, over indulgence in soporifics such as the barbituric acid compounds is treated very briefly. Some of the chapters most interesting to the reviewer concern habituations to less well known drugs such as mescaline, kava-kava, certain toadstools, and betel. In the treatment of each drug attention is given to its source, its method of preparation for use, its physical and chemical properties, an outline of its pharmacologic action, an estimate of the extent of its use, and its sociologic importance as a habit forming or addicting drug.

The author's definitions of habituation and addiction do not conform with those in use in this country at the present time, but this is of minor importance since his meaning of the terms is made clear. The content suffers to some extent in translation from the German by the use of unusual terminology and in some cases idiomatic construction. Probably a more serious criticism from the viewpoint of the interest of the American reader is that discussions of the extent of the use of various drugs and laws controlling drug traffic refer to Europe and particularly to Germany with only incidental reference to conditions in this country. Also bibliographic references are drawn chiefly from the German literature.

Although a comprehensive treatment of the pharmacology of each drug is unnecessary in a book of this type numerous statements are made and impressions given to which exception should be taken. In discussing the chronic effects of alcohol no reference is made to the part played by avitaminosis. The impression is given that cirrhosis of the liver may be produced by alcohol in experimental animals. References to the chemistry of the active principle of hashish indicate unfamiliarity with recent developments in this field. Also in numerous instances a positive stand is taken where the scientific evidence, is at best, controversial.

As stated in the preface, the purpose of the book is in part to combat drug addiction and habituation by the dissemination of knowledge concerning the dangers involved. In pursuit of this worthy purpose the author frequently gives greater credence to evidence of the deleterious effects of the drugs rather than to contrary reports when the literature on the subject is controversial. Although the reader will find much of interest it must be recognized that a thesis rather than a purely scientific consideration of the subject has been presented.

Ralph G. Smith, M. D.

Current Therapies of Personality Disorders: By Bernard Glueck, M. D. New York, Grune & Stratton, 1946. Pp. 296.

The results of the proceedings of the thirty-fourth annual meeting of the American Psychopathological Association, are available now in this volume to guide physicians in management and treatment of personality disorders. Twenty experts present a series of timely papers devoted to the modern psychopathic hospital, psychochemical technics, phychiatric guidance and rehabilitation. There is invaluable material for neuropsychiatrists that will help to rid personality disorders of the mystery clouding their proper medical recognition. You will come from reading this book with an enlarged conception of the human organism as an almost infinite complexity of action and reaction between structure, normal and abnormal, and function, orderly and disordered. It can be recommended to those who desire a comprehensive survey of the subject. It should present considerable food for thought to therapists in the field of human personality and human relations.

C. P. May, M. D.

Preoperative and Postoperative Treatment: Edited by Lt. Col. Robert L. Mason, M. C., A. U. S., and Harold A. Zintel, M. D. 2nd ed. Philadelphia, W. B. Saunders & Co. 1946. Pp. 584. Price $7.00.

This volume fills a real need. It should be on the shelf of every hospital library. It should be a

welcome addition to the reference library of surgeons. It is an invaluable reference book for the resident staff.

Each chapter in this volume gives evidence of careful preparation. The material is as near the current literature as is possible for a printed volume to be. It is not often that one can say without hesitation — this is a volume which deserves a place in any man's library.

ISIDORE COHN.

Textbook of Gynecology: By Arthur Hale Curtis, M. D. 5th ed. Philadelphia, W. B. Saunders, 1946. Illus. Pp. 755. Price $8.00.

This new edition has 455 illustrations and 755 pages. It has some new additions on almost every gynecologic subject, and numerous revisions. The new additions to the chapter on Other Tumors of the Uterus are nicely presented and well illustrated with photographic plates. The principal revisions noted are in endocrinopathic disturbances and ovarian tumors.

The illustrations and material from the fourth edition have been used and 54 new illustrations added. The general plan of presentation is the same.

This new book is an improvement over the fourth edition and is an excellent reference book for clinicians and students. It is very well written and illustrated, and presents recent trends in physiology, pathology and treatment in gynecologic disease.

LEE H. LORENZEN, M. D.

Bibliography of Infantile Paralysis, 1789-1944, with selected abstracts and annotations, prepared under the direction of the National Foundation for Infantile Paralysis, Inc.: Edited by Morris Fishbein, M. D., comp. by Ludvig Hektoen, M. D. and E. M. Salmonsen. Philadelphia, J. B. Lippincott, 1946. Pp. 672. Price $15.00.

Of widespread interest among neuropsychiatrists and orthopedists, should be this bibliography with abstracts from the literature, on infantile paralysis. It includes a comprehensive list of periodicals covering the clinical and investigative work on the subject and its phases since its description by Underwood in 1789.

The elements are arranged chronologically and access to the various aspects of the condition is facilitated by an extensive, complete and thorough index covering the references and articles by number. This method of arrangement has been found the most convenient of any of the various technics that are used in the preparation for publication of material of this kind. A vast amount of valuable data is thus made available.

C. P. MAY, M. D.

PUBLICATIONS RECEIVED

Grune & Stratton, New York: Mongolism and Cretinism, by Clemens E. Bends, M. D.

Lea & Febiger, Philadelphia: Principles of Hematology, by Russell L. Haden, M. A., M. D.

J. P. Lippincott, Philadelphia: Medical Uses of Soap, by Morris Fishbein, M. D.

Russell Sage Foundation, New York: Music in Hospitals, by Willem Van De Wall.

W. B. Saunders, Philadelphia: Practical Malariology, by Paul F. Fussell, M. D., M. P. H., Luther S. West, Ph. D., Reginald D. Manwell, Sc. D.

Charles C. Thomas, Publisher, Springfield, Ill.: Harvey Cushing, A Biography, by John F. Fulton. Principles in Roentgen Study of the Chest, by William Snow, M. D.

Walker, Evans & Cogswell Co., Charleston, S. C.: Radium Lost and Found, by Robert B. Taft, M. D., B. S., M. A., F. A. C. R.